GASTROENTEROLOGY

ABSTRACTS AND CITATIONS

NATIONAL INSTITUTE OF ARTHRITIS, METABOLISM, AND DIGESTIVE DISEASES

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PREFACE

GASTROENTEROLOGY ABSTRACTS and CITATIONS is a publication of the National Institute of Arthritis, Metabolism and Digestive Diseases. This specialized information medium has been initiated to fill an existing great need in the field of gastroenterology and to assist the Institute in meeting its obligations to foster and support laboratory and clinical research into the nature, causes, and therapy of diseases of the gastrointestinal tract. Publication GASTROENTEROLOGY ABSTRACTS and CITATIONS makes available citations of all current papers relevant to this field from medical journals published throughout the world. Approximately one-third of the citations dealing with the major aspects of gastroenterology have accompanying abstracts.

The issuing of GASTROENTEROLOGY ABSTRACTS and CITATIONS under the auspices of the National Institute of Arthritis, Metabolism, and Digestive Diseases will provide a much needed current awareness tool to scientists and will facilitate greater integration of research and clinical efforts in this field. The number and great diversity of publications in the area of gastroenterology makes it imperative that an appropriate service be available to investigators and practitioners so that they may be apprised of progress with a minimum of delay. Our aim is to provide the readers with a readily systematized compilation of current published work. The publication will provide the greatest usefulness if these interested investigators will contribute their ideas and comments for consideration whenever possible.

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LANGUAGE ABBREVIATIONS

Afrikaans	Afr.	Georgian	Geo.	Persian	Per.
Albanian	Alb.	German	Ger.	Polish	Pol.
Arabic	Ara.	Greek	Gre.	Portuguese	Por.
Armenian	Arm.	Hausa	Hau.	Pushto	Pus.
Azerbaijani	Aze.	Hebrew	Heb.	Rumanian	Rum.
Belorussian	Bel.	Hindi	Hin.	Russian	Rus.
Bengali	Ben.	Hungarian	Hun.	Serbian	Ser.
Bulgarian	Bul.	Icelandic	Ice.	Slovak	Slo.
Burmese	Bur.	Indonesian	Ind.	Slovene	Slv.
Chinese	Chi.	Interlingua	Int.	Spanish	Spa.
Croatian	Cro.	Italian	Ita.	Swahili	Swa.
Czechoslovakian	Cze.	Japanese	Jpn.	Swedish	Swe.
Danish	Dan.	Kirghiz	Kir.	Tagalog	Tag.
Dutch	Dut.	Korean	Kor.	Tamil	Tam.
English	Eng.	Latin	Lat.	Telugu	Tel.
Esperanto	Esp.	Latvian	Lav.	Thai	Tha.
Estonian	Est.	Lithuanian	Lit.	Turkish	Tur.
Finnish	Fin.	Marathi	Mar.	Ukranian	Ukr.
Flemish	Fle.	Masai	Mas.	Urdu	Urd.
French	Fre.	Multilingual	Mlt.	Vietnamese	Vie.
Gaelic	Gae.	Norwegian	Nor.	Welsh	Wel.

ABBREVIATIONS USED IN ABSTRACTS

ACTH	adrenocorticotropic hormone	ml, µl	milli-, microliter(s)
ADP	adenosine diphosphate	mm	millimeter(s)
AMP	adenosine monophosphate	mOsm	milliosmolar; milliosmeter(s)
amp	ampere	msec	millisecond(s)
ATP	adenosine triphosphate	mV	millivolt(s)
A	angstrom	min	minute(s)
BSP	sulfobromophthalein	MTD	maximum tolerated dose
C	degrees centigrade	NAD	nicotinamide adenine dinucleotide
cal	calories	NADH	reduced nicotinamide adenine dinucleotide
cc	cubic centimeter		
cm	centimeter(s)	NADP	nicotinamide adenine dinucleotidephosphate
dl	deciliter		
DNA	deoxyribonucleic acid	NADPH	reduced nicotinamide adenine dinucleotidephosphate
ED_{50}	median effective dose(s)		
EDTA	ethylenediamine tetraacetic acid	ng	nanogram (10^{-9})
e.g.	for example	nm	nanometer(s)
EEG	electroencephalogram	Pa	ambient pressure
ESR	erythrocyte sedimentation rate	PAS	periodic acid - Schiff
g	gram(s)	pg	picogram (10^{-12})
µg	microgram(s)	pgEq	picogram equivalent(s)
Hz	Hertz	p.o.	orally
hr	hour(s)	ppb	parts per billion
Ig	immunoglobulin(s)	ppm	parts per million
i.m.	intramuscular	r	Roentgen
i.p.	intraperitoneal	RBC	red blood cells (erythrocytes), red blood count
IU	international unit(s)		
i.v.	intravenous	resp.	respectively
kcal	kilocalorie(s)	RNA	ribonucleic acid
kg	kilogram(s)	s.c.	subcutaneous
K_m	Michaelis constant	sec	second(s)
LD_{50}	median lethal dose(s)	SGOT	serum glutamic-oxalacetic transaminase
LDH	lactic acid dehydrogenase		
m	meter(s)	SGPT	serum glutamic-pyruvic transaminase
M	molar; mole(s)		
mM	millimolar; millimole(s)	U	unit(s)
µM	micromolar; micromole(s)	UV	ultraviolet
ma	milliampere(s)	V	volt
mCi, µCi	milli-, microcurie(s)	WBC	white blood cells (leukocytes), white blood count
mEq	milliequivalent(s)		
mg	milligram(s)	yr	year(s)

PRE-CLINICAL SCIENCES

MORPHOLOGY, CELLULAR STRUCTURE AND HISTOCHEMISTRY

8620 CHARACTERIZATION OF COLUMNAR ABSORPTIVE EPITHELIAL CELLS ISOLATED FROM RAT JEJUNUM. (Eng.) Towler, C. M.; Pugh-Humphreys, G. P.; Porteous, J. W. (Dept. Biochemistry, Univ. Aberdeen, Aberdeen AB9 1AS, Scotland). *J. Cell Sci.* 29:53-75; 1978.

A comprehensive qualitative and quantitative assessment of the origin, the yield, and the biochemical integrity of cells isolated from rat jejunum was carried out. Cell isolation yielded cells that were nearly all structurally intact, that represented the whole of the population of cells originally present on the walls of the villi of rat jejunum, and that were almost all columnar absorptive cells; the only other cells present (in small numbers) were goblet cells and WBC. One possible reservation about the integrity of the isolated columnar absorptive cells arose from the observation that some cells contained many more vacuoles, especially in the supranuclear region, than would be expected from examination of sections of similar cells *in situ* in the intact intestine. The isolated cells retained a number of solutes (orthophosphate, ATP, ADP, AMP, lactate) at intracellular concentrations exceeding those of the solutes in the incubation medium; each of these test solutes would be expected to pre-exist in the isolated cells or to be generated intracellularly during incubation. A number of solutes (α-methyl-D-glucoside, 3-*O*-methyl-D-glucose, D-galactose, L-valine, L-leucine) were translocated from the incubation medium into the cell and accumulated there at a concentration exceeding that found in the medium. Two inhibitors of fatty acid catabolism (2-bromo-hexanoic acid and 4-pentenoic acid) failed to prevent intracellular accumulation of α-methyl-D-glucoside, possibly indicating that the accumulation process is independent of fatty acid catabolism or that these inhibitors failed to penetrate the isolated cells. All other inhibitors tested (ouabain, cyanide, dinitrophenol, phlorhidzin, and carbonyl-cyanide-*m*-chlorophenyl-hydrazine) had the same effects on α-methyl-D-glucoside accumulation as would be expected from observations with more complex preparations of intestine. The failure of ouabain to give complete inhibition of intracellular methylglucoside accumulation suggested that these cells contain an ouabain-insensitive Na^+-dependent ATPase similar to that found in other tissues. Isolated cells respired at a steady rate (18.6 ± 0.9 μmol O_2/hr/mg cell DNA) using endogenous substrates; the addition of 5 mM L-glutamine stimulated respiration (to 25 μmol O_2/hr/mg cell DNA), supporting the previous suggestion that plasma glutamine might be an important respiratory substrate for intestine. The addition of D-glucose stimulated respiration and gave rise to increased lactic acid production. Extracellular L-valine was incorporated into cell protein. Intracellular ATP, ADP, and AMP levels could be held constant, or changed in a predictable way. The observations suggest that isolated and incubated cells could be valuable for the further exploration of the biochemical functions of the small intestine.

8621 ULTRASTRUCTURAL MORPHOMETRIC INVESTIGATIONS ON NORMAL HUMAN LIVER BIOPSIES. (Eng.) Roessner, A.; Kolde, G.; Stahl, K.; Blanke, G.; van Husen, N.; Themann, H. (Lehrstuhl fur Medizinische Cytobiologie der Universitat, Westring 3, 4400 Munster, W. Germany). *Acta Hepatogastroenterol.* 25(2):119-123; 1978.

Morphometric ultrastructural evaluations of liver needle biopsies from 14 normal human subjects were carried out to obtain baseline data for comparative purposes and to determine whether such evaluations yield reliable results in spite of the higher range of distribution in this material compared with experimental liver material. The data were related to one of two reference systems: (1) unit volume of liver tissue or (2) unit volume of hepatocytes. Liver tissue volume was subdivided into sinusoid volume (0.211 ± 0.025 ml/ml liver tissue), hepatocyte volume (0.785 ± 0.025 ml/ml), and bile canalicular volume (0.0043 ± 0.0005 ml/ml). The hepatocyte cytoplasm accounted for most of the hepatocyte volume (0.729 ± 0.025 ml/ml), while the number of hepatocyte nuclei was 123.02 ± 8.20 x 10^6/ml liver tissue. The surface area of bile canaliculi was estimated to be 0.0508 ± 0.0042 m^2/ml liver tissue (a minimum estimate). The hepatocyte volume contained 0.076 ± 0.003 ml/ml nuclei, 0.924 ± 0.003 ml/ml cytoplasm, 0.138 ± 0.005 ml/ml mitochondria, 0.0099 ± 0.0006 ml/ml microbodies, 0.0045 ± 0.0013 ml/ml fat inclusions, and 0.0056 ± 0.0017 ml/ml lysosomes. There were 201 ± 14 x 10^9 mitochondria/ml hepatocytes (with inner and outer mitochondrial membrane surface areas of 2.171 ± 0.134 and 1.162 ± 0.056 m^2/ml hepatocytes, resp.) and 55.49 ± 2.39 x 10^9 microbodies/ml hepatocytes. The surface area of rough and smooth endoplasmic reticulum was 1.713 ± 0.140 and 1.981 ± 0.003 m^2/ml hepatocytes. The standard error of nearly all tested parameters was less than 10% of the mean, indicating that ultrastructural morphometric evaluations of normal human liver biopsy samples yield reliable results.

8622 LIGHT AND ELECTRON MICROSCOPIC HISTOCHEMISTRY OF THE SEROUS SECRETORY GRANULES IN THE SALIVARY GLANDULAR CELLS OF THE MONGOLIAN GERBIL (Mongolian meridianus) AND RHESUS MONKEY (Macaca irus). (Eng.) Ichikawa, M.; Ichikawa, A. (Yokohama City Univ. Sch. Medicine, Yokohama 232, Japan). *Anat. Rec.* 189(1):125-139; 1977.

8623 MORPHOLOGY OF RESTING AND STIMULATED DISPERSED OXYNTIC CELLS. (Eng.) Lechago, J. (Harbor General Hosp., 1000 W. Carson St., Torrance, CA 90509). *Gastroenterology* 73(4, Part 2): 958; 1977.

8624 CURRENT CONCEPTS OF LIVER STRUCTURE AS RELATED TO FUNCTION. (Eng.) Jones, A. L.; Schmucker, D. L. (Veterans Admin. Hosp., San

Francisco, CA). *Gastroenterology* 73(4, Part 1): 833-851; 1977.

8625 MORPHOLOGY OF THE GUT ASSOCIATED LYMPHOID SYSTEM IN HEALTH AND DISEASE: A REVIEW. (Eng.) Whitehead, R.; Skinner, J. M. (Flinders Medical Center, Bedford Park, South Australia 5042, Australia). *Pathology* 10(1):3-16; 1978.

8626 LIGHT AND SCANNING ELECTRON MICROSCOPIC OBSERVATIONS OF THE EFFECTS OF SUBLETHAL DOSES OF METHOTREXATE ON THE RAT SMALL INTESTINE. (Eng.) Jeynes, B. J.; Altmann, G. G. (Health Sciences Center, Univ. Western Ontario, London, Ontario N6A 5C1, Canada). *Anat. Rec.* 191(1):1-17; 1978.

8627 MOTOR INNERVATION OF THE STRIATED OESOPHAGUS MUSCLE: PART 1. INTRAMURAL DISTRIBUTION OF THE RIGHT AND LEFT VAGUS NERVE IN THE RAT OESOPHAGUS AS REVEALED BY THE GLYCOGEN DEPLETION TECHNIQUE. (Eng.) Gruber, H. (II. Anatomisches Institut der Universitat Wien, A-1090 Vienna, Austria). *J. Neurol. Sci.* 36(1):41-53; 1978.

8628 THE THREE-DIMENSIONAL FINE STRUCTURE OF THE LIVER AS REVEALED BY SCANNING ELECTRON MICROSCOPY. (Eng.) Motta, P. M. (Faculty Medicine, Univ. Rome, Rome, Italy). *Int. Rev. Cytol.* (Suppl. 6):347-399; 1977.

8629 STRUCTURE AND FUNCTION OF GUT ENDOCRINE CELLS. (Eng.) Fujita, T.; Kobayashi, S. (Niigata Univ. Sch. Medicine, Niigata, Japan). *Int. Rev. Cytol.* (Suupl. 6):187-233; 1977.

See also, 8665, 8680, 8720, 8745, 8761, 8773, 8823, 9127, 9205.

ABSORPTION - EXCRETION - TRANSPORT

8630 SIMILARITY IN EFFECTS OF Na^+ GRADIENTS AND MEMBRANE POTENTIALS ON D-GLUCOSE TRANSPORT BY, AND PHLORIZIN BINDING TO, VESICLES DERIVED FROM BRUSH BORDERS OF RABBIT INTESTINAL MUCOSAL CELLS. (Eng.) Toggenburger, G.; Kessler, M.; Rothstein, A.; Semenza, G.; Tannenbaum, C. (Laboratorium fur Biochemie, Eidgenossische Technische Hochschule, Universitatsstrasse 16, CH 8092 Zurich, Switzerland). *J. Membr. Biol.* 40(3):269-290; 1978.

To elucidate the role of Na^+, Na^+ gradients, electrical potentials, and anion substitution on sugar transport and on phlorizin binding, kinetic studies were conducted in vesicles derived from brush borders of rabbit intestinal mucosal cells. Phlorizin (20 and 100 mM) strongly inhibited D-glucose (30 μM) uptake by the vesicles under a 100 mM inward NaSCN gradient. Under equilibrium conditions, a large amount of low affinity phlorizin binding to intestinal brush border vesicles was observed, but there was no evidence of a high affinity component that could be correlated with the high inhibitory potency of the phlorizin. In the presence of a transmembrane NaSCN gradient, the rapid high affinity binding of phlorizin was observed within 1 sec. D-glucose abolished the high affinity component to the extent of about 80%; low affinity binding was not significantly reduced by D-glucose. Phlorizin binding was markedly reduced by substitution of NaCl for NaSCN or by allowing the vesicles to equilibrate in NaSCN for 1 hr before the addition of phlorizin. Phlorizin binding was increased two-fold by a 90 mM choline thiocyanate gradient in the absence of a Na^+ gradient. However, in the absence of Na^+, no increase in D-glucose transport or in phlorizin binding was observed. The involvement of the small intestinal Na^+-dependent monosaccharide transporting system in the high affinity phlorizin binding was demonstrated and phlorizin was shown to be a fully competitive inhibitor of the monosaccharide transport process. The effect of the potential gradient ($\Delta\psi$), studied independent of an inward Na^+ gradient, showed $\Delta\psi$ to be a necessary factor in determining high affinity glucose-protectable phlorizin binding, but Na^+ must also be present. Furthermore, Na^+ alone without $\Delta\psi$ does not produce optimal high affinity glucose-protectable phlorizin binding. Three models attempting to integrate $\Delta\psi$ and Na^+-dependence of high affinity phlorizin binding with the mechanism of Na-dependent monosaccharide transport are proposed. The available experimental evidence does not favor any one of these models. The complex nature of the phlorizin dissociation function was shown was well as the importance of the

experimental conditions in its determination. Both the presence of Na^+ and of an electrical transmembrane potential difference are required for optimal D-glucose-protectable phlorizin binding to brush border membranes from rabbit small intestine. Phlorizin is bound to the D-glucose transporting protein.

8631 EFFECT OF ANIONS ON AMILORIDE-SENSITIVE, ACTIVE SODIUM TRANSPORT ACROSS RABBIT COLON, IN VITRO: EVIDENCE FOR "TRANS-INHIBITION" OF THE Na ENTRY MECHANISM. (Eng.) Turnheim, K.; Frizzel, R. A.; Schultz, S. C. (Univ. Pittsburgh, Sch. Medicine, Pittsburgh, PA 15261). *J. Membr. Biol.* 37(1):63-84; 1977.

The effects of replacing Cl with sulfate, isethionate, hydroxypropane-sulfonate (HPSA), ethane-sulfonate (ESA), or *p*-phenolsulfonate (PSA) on active transport (J^{Na}_{net}) were investigated in strips of rabbit descending colon. Replacement of 107 mM Cl in the bathing solutions with isoosmolar sulfate, isethionate, HPSA and, to a lesser degree, ESA stimulated J^{Na}_{net} when the basolateral pump mechanism was not saturated. These effects were rapid in onset and were readily reversible. These stimulatory anions decreased the resistance of the amiloride-sensitive Na entry step at the mucosal membrane. However, when the active Na pump mechanism at the basolateral membrane was saturated, the stimulatory anions did not decrease the resistance of the Na entry process. These findings suggest the presence of a negative feedback system between the activity of the pump mechanism and the resistance of the Na entry step, which may be mediated by the size of the intracellular Na transport pool. In other words, it seems that when the basolateral pump is operating at its maximal rate, the resistance to Na entry across the mucosal membrane through the amiloride-sensitive pathway is at a minimum and cannot be further decreased.

8632 REVERSIBLE INHIBITION OF SODIUM ABSORPTION IN THE RAT COLON BY LOW pH: A MECHANISM FOR DIARRHEA. (Eng.) Chung, R. S.; Johnson, G. M. (Veterans Admin. Hosp., Iowa City, IA). *Surg. Forum* 28:435-437; 1977.

The effect of acidic pH on ion transport *in vivo* was studied in male Sprague-Dawley rats in which 10 cm of proximal colon were cannulated and perfused after the rats had been injected with ketamine (40 mg/kg, i.m.). The pH of the perfusate was 6.7-7.1 (control), 4.5, 3.0, 2.3, or 2.0. Net ion fluxes were measured. H^+ disappearance was linearly related to luminal pH ($r=0.942$, $p<0.001$). The net absorption of Na^+ and Cl^- and the volume absorption observed in the controls was decreased and reversed by decreasing pH; net secretion of Na^+ was observed below pH 3 ($p<0.01$), net secretion of Cl^- began between pH 3.0 and pH 2.3, and net volume secretion was observed below pH 4.5 ($p<0.01$). Histological studies showed that structural damage of superficial cells occurred with increasing severity as pH decreased, beginning at pH 3. The initial potential difference (PD) was greater at lower pH; PD decreased slowly during all experiments, with larger decreases at lower pH. It is suggested that PD may account for the major portion of H^+ movement, that net Na^+ movement may occur passively by paracellular shunt pathways, or that a much lower pH is required to affect Na^+ cellular transport, and that Na^+ and volume absorption changes driven by reduced pH may play a role in the diarrhea seen in clinical states of reduced pH in the colonic lumen.

8633 ABSORPTION OF PANCREATIC LIPASE FROM THE DUODENUM INTO LYMPHATICS. (Eng.) Papp, M.; Feher, S.; Folly, G.; Horvath, E. J. (Hungarian Acad. Sciences, P.O. Box 67, H-1450 Budapest, Hungary). *Experientia* 33(9):1191-1192; 1977.

To study the absorption of pancreatic enzymes, lipase activity was determined in the duodenal lymph and in the blood plasma of the femoral artery of dogs prior to and during maximal hormonal stimulation of exocrine pancreatic function. Duodenal lymph was collected in anesthetized dogs first for 90 min during saline infusion and then for 120 min during and after stimulation of exocrine pancreatic secretion by the i.v. infusion of 4 U/kg secretin and pancreozymin. Arterial blood samples were collected before, at 20, 40, and 60 min during stimulation, and at 90 and 120 min after stimulation. The mean basal pancreatic lipase activity was significantly higher in the duodenal lymph than in arterial blood plasma ($p<0.01$). When hormonally stimulated pancreatic juice was secreted into the duodenum, lipase activity was significantly elevated both in the duodenal lymph ($p<0.01$) and in each of the plasma samples ($p<0.01$) as compared with the initial values. Under these conditions, the mean pancreatic lipase activity in the duodenal lymph samples was significantly higher ($p<0.01$) than in the plasma samples. These results are thought to reflect pancreatic lipase absorption by the duodenum into lymphatics.

8634 DIPEPTIDE TRANSPORT IN THE INTESTINAL MUCOSA OF DEVELOPING RABBITS. (Eng.) Rubino, A.; Guandalini, S. (Second Sch. Medicine, Naples, Italy). *Ciba Found. Symp.* 50(New Series): 61-77; 1977.

The transport and selective carrier mechanisms of two dipeptides, glycyl-L-proline (G-pr) and glycyl-L-phenylalanine (G-ph), across the small intestinal brush border were studied in fetal, newborn, and suckling rabbits. G-pr is transported intact across the brush border, while G-ph is hydrolyzed at the border and the amino acids then transported across the brush border. Using glycine-labeled G-pr and phenylalanine-labeled G-ph, the intestinal uptake of each substrate was calculated from the amount of radioactivity in the tissue at the end of a 60-sec incubation period and compared with the uptake of the corresponding free acid. No significant differences in the uptake of free glycine in either ileum or jejunum were observed, but G-pr uptake showed a striking perinatal

peak with a gradual increase from the 25th day of gestation, remained constant during the first 6 postnatal days, and then declined to reach adult values at 50 days after birth. G-pr transport proceeded by a single-carrier mechanism whose rate was stimulated by sodium ions. This transport did not appear to differ fundamentally from the influx process in adult rabbits, except that the maximal velocity was much larger in newborns than in adults (11.2 ± 2.5 and 0.81 ± 0.25 μmol/cm^2/hr, resp.). Affinity constants for G-pr uptake were 0.95 ± 0.26 and 0.91 ± 0.35 mM, resp., for newborns and adults. The two components of G-ph uptake were measured separately using a 40-fold excess of either G-pr or leucine as selective inhibitors of the intact peptide transport route and of the free acid transport after hydrolysis route, resp. Again, differences were noted between the uptake of free and dipeptide-bound phenylalanine. It was postulated that in fetuses the hydrolytic enzyme might not have developed and that it might develop completely only after birth. In newborn rabbits, the uptake of free amino acids was larger in the ileum, while the uptake of dipeptides was larger in the jejunum. Additional evidence of the distinction between the dipeptide and neutral amino acid transport processes was obtained in neonate rabbits; differences in the absorption of dipeptides and free amino acids along the length of the small bowel and a larger maximal velocity of the dipeptide carrier mechanism were observed in newborn rabbits. The findings may have significance in determining elemental diets for premature and full-term infants.

8635 EFFECTS OF INTRADUODENAL ACID ON GALLBLADDER NET WATER ABSORPTION AND MOTILITY IN THE CAT. (Eng.) Jansson, R. (Sahlgrenska sjukhuset, S-413 45 Goteborg, Sweden). *Scand. J. Gastroenterol.* 13(2):209-215; 1978.

Gallbladder absorption and motility were studied in anesthetized cats during the stimulation of gastrointestinal hormone release by intraduodenal acid infusion. Gallbladder function was studied with a perfusion technique that allows absorption and motility to be monitored simultaneously. The gallbladder is perfused at a constant rate from neck to fundus, and net water absorption is registered as a continuous decrease in weight on an electromagnetic balance. Hepatic bile flow is registered with a drop recorder. When the gallbladder was perfused by hepatic bile, the average net water absorption in the gallbladder was reduced from 0.60 ml/hr during the control period to 0.18 ml/hr ($p<0.01$) during HCl infusion (0.8 mmol/kg/hr). When the gallbladder was perfused by saline, intraduodenal acid infusion reduced net water transport from 1.34 ml/hr to 0.60 ml/hr ($p<0.01$). Acid infusion evoked a contraction of the gallbladder which, when estimated, expelled one-fifth of its contents. Bile flow increased from 1.37 ± 0.12 to 2.14 ± 0.16 ml/hr during acid instillation, and pancreatic secretion increased from 0 to 3.86 ± 0.77 ml/hr. The duodenal instillation of isotonic bicarbonate solution caused no changes in gallbladder net water transport, motility, or in bile or pancreatic secretion. A comparison of the present results with a study on the effects of i.v. secretin on gallbladder function suggests that the reduction in gallbladder net water absorption in response to intraduodenal acid can be explained by endogenously released secretin; the amount of secretin release evoked by acid infusion in this study can be assumed to be less than 2 U/kg/hr, since this amount reduced and even abolished absorption in the previous study. Gallbladder contraction observed after acid infusion may be explained by an endogenous release of cholecystokinin.

8636 ENHANCED NON-ELECTROLYTE UPTAKE IN VITRO BY THE DOG JEJUNAL MUCOSA IN THE CHOLERA-TOXIN-INDUCED SECRETORY PHASE. (Eng.) Robinson, J. W. L.; Mirkovitch, V.; Sepulveda, F. V.; Menge, H. (Centre Hospitalier Universitaire Vaudois, 1011 Lausanne, Switzerland). *J. Physiol. (Paris)* 73(1): 5-12; 1977.

8637 GLUCOSE AND CATION TRANSPORT IN RAT JEJUNUM, ILEUM AND COLON IN VIVO: EFFECTS OF ANIONIC AND NONIONIC SURFACTANTS, AND OF DESOXYCHOLATE. (Eng.) Sund, R. B.; Matheson, I. (Inst. Pharmacy, Univ. Oslo, P.O. Box 1068, Blindern, Oslo 3, Norway). *Acta Pharmacol. Toxicol.* 42(5):253-258; 1978.

8638 GENERALITY IN EFFECTS OF TRANSMUCOSAL FLUID MOVEMENT AND GLUCOSE ON DRUG ABSORPTION FROM THE RAT SMALL INTESTINE. (Eng.) Kitazawa, S.; Ito, H.; Johno, I.; Takahashi, T.; Takenaka, H. (Kyoto Univ. Hosp., Kawara-cho, Shogoin, Sakyo-ku, Kyoto 606, Japan). *Chem. Pharm. Bull.* 26(3):915-924; 1978.

8639 ROLE OF INTESTINAL MUCUS IN THE ABSORPTION OF QUININE AND WATER-SOLUBLE DYES FROM THE RAT SMALL INTESTINE. (Eng.) Nakamura, J.; Shima, K.; Kimura, T.; Muranishi, S.; Sezaki, H. (Faculty Pharmaceutical Sciences, Kyoto Univ., Yoshida Shimoadachi-cho, Sakyo-ku, Kyoto, Japan). *Chem. Pharm. Bull.* 26(3):857-863; 1978.

8640 EFFECTS OF FREE FATTY ACIDS AS MEMBRANE COMPONENTS ON PERMEABILITY OF DRUGS ACROSS BILAYER LIPID MEMBRANES: A MECHANISM FOR INTESTINAL ABSORPTION OF ACIDIC DRUGS. (Eng.) Hori, R.; Kagimoto, Y.; Kamiya, K.; Inui, K. I. (Hiroshima Univ. Sch. Medicine, Kasumi 1-2-3, Hiroshima 734, Japan). *Biochim. Biophys. Acta* 509(3):510-518; 1978.

8641 EFFECT OF LOW CALCIUM AND LOW PHOSPHORUS DIETS ON THE INTESTINAL ABSORPTION OF PHOSPHATE IN INTACT AND PARATHYROIDECTOMIZED PIGS. (Eng.) Fox, J.; Care, A. D. (Dept. Animal Physiology and Nutrition, Univ. Leeds, Leeds LS2 9JT, England). *J. Endocrinol.* 77(2):225-231; 1978.

8642 THE EFFECT OF INORGANIC PHOSPHATE ON INTESTINAL ABSORPTION AND URINARY EXCRE-

TION OF CALCIUM IN MAN. (Eng.) Falch, J. A.; Berstad, A.; Jorgensen, H. (Aker Hosp., Oslo, Norway). *J. Oslo City Hosp.* 27(9):105-110; 1977.

8643 MOVEMENT OF WATER AND SODIUM DURING THE ABSORPTION OF GLUCOSE AND MALTOSE IN THE JEJUNUM IN MAN [Abstract]. (Eng.) Holmes, R.; Lobley, R. W.; Sandle, G. I. (Royal Infirmary, Manchester, England). *J. Physiol. (Lond.)* 276:53P-54P; 1978.

8644 THE ABSORPTIVE VIABILITY OF ISOLATED INTESTINE PREPARED FROM DEAD ANIMALS. (Eng.) Gardner, M. L. G. (Univ. Edinburgh Medical Sch., Teviot Place, Edinburgh EH8 9AG, England). *Q. J. Exp. Physiol.* 63(1):93-95; 1978.

8645 MUCOSAL TRANSFERRIN AND FERRITIN FACTORS IN THE REGULATION OF IRON ABSORPTION. (Eng.) El-Shobaki, F. A.; Rummel, W. (Nutrition Dept., Natl. Res. Center, Dokki, Cairo, Egypt). *Res. Exp. Med.* 171(3):243-253; 1977.

8646 POSSIBLE ROLE OF INTESTINAL ALKALINE PHOSPHATASE ACTIVITY IN THIAMINE TRANSPORT. (Eng.) Matsuda, T.; Baba, A.; Iwata, H. (Dept. Pharmacology, Osaka Univ., 133-1, Yamada-kami, Suita-shi, Osaka, Japan). *Experientia* 34(1):18-20; 1978.

8647 SALIVARY GLAND K^+ TRANSPORT: IN VIVO STUDIES WITH K^+-SPECIFIC MICROELECTRODES. (Eng.) Poulsen, J. H.; Bledsoe, S. W. (Univ. California Sch. Medicine, San Francisco, CA 94143). *Am. J. Physiol.* 234(1):E79-E83; 1978.

See also, 8758.

MOTILITY

8648 VAGAL INFLUENCE ON DUODENAL MOTOR ACTIVITY. (Eng.) Mir, S. S.; Mason, G. R.; Ormsbee, H. S., III (Univ. Maryland Sch. Medicine, Baltimore, MD). *Am. J. Surg.* 135(1):97-101; 1978.

The extrinsic and intrinsic vagal influences on duodenal motor activity were studied in the anesthetized dog to determine whether electrical vagal stimulation influences duodenal motility specifically via extrinsic fibers to the duodenum or through an intrinsic innervation that crosses the gastroduodenal junction. Dogs were vagotomized, and stimulating electrodes were placed on the distal cut ends of the vagal trunks. After truncal vagal stimulation at 32 Hz, 5 msec, and 3 mamp, the gastric antrum and the duodenum at 3 cm and 8 cm demonstrated significant increases in contractile activity ($p<0.001$ and $p<0.01$, resp.). Upon termination of the stimulatory current, poststimulatory contractions, or off responses, were frequently noted on the antrum and the duodenum. Significant poststimulatory activity was observed on the antrum in all phases of the experiment. After truncal vagotomy followed by extrinsic denervation of the duodenum (DENER), vagal stimulation produced an increase in motility index at the antrum ($p<0.001$) and at both sites in the duodenum ($p<0.01$). Off responses were still demonstrable early in the poststimulatory period in both organs. After truncal vagotomy, followed by DENER and then by pyloric transection (PT), vagal stimulation continued to produce an increased motility index at the antrum ($p<0.01$), but no increase in motor activity was seen in the duodenum. Off responses no longer appeared at either duodenal position upon termination of the stimulus current. After truncal vagotomy and PT, stimulus-induced contractions on the antrum and duodenum were still present. However, when truncal vagotomy was followed by PT and then by DENER, only the antrum responded with increased motor activity. These results support the concept that the motor innervation to the duodenum is composed of extrinsic vagal fibers and intrinsic vagal nerves that appear to first enter the antrum and then cross the gastroduodenal junction to innervate the duodenum.

8649 POTENTIATION BY DIPYRIDAMOLE OF THE INHIBITION OF GUINEA-PIG ILEUM TWITCH RESPONSE CAUSED BY ADENINE DERIVATIVES. (Eng.) Moritoki, H.; Kanbe, T.; Maruoka, M.; Ohara, M.; Ishida, Y. (Faculty Pharmaceutical Sciences, Univ. Tokushima, Shomachi-1, Tokushima 770, Japan). *J. Pharmacol. Exp. Ther.* 204(2):343-350; 1978.

The inhibitory actions of adenine derivatives on transmural-stimulated ileal contractions were studied in the guinea pig, and the potentiation of the effects of these derivatives by dipyridamole was investigated. Adenosine and adenine nucleotides caused dose-dependent inhibition of ileal

contraction induced by transmural stimulation (twitch response) at 0.1 Hz. The inhibitory effects of the adenine derivatives were potentiated by pretreatment with dipyridamole at concentrations of over 10^{-8} M for 30 min. Dipyridamole shifted the dose-response curve for adenosine to the left with concentrations of 3×10^{-8} and 3×10^{-7} M dipyridamole causing about 8- and 13-fold shifts, resp. Tritium activity, which was accumulated during incubation of the ileal segments with ^{3}H-adenosine, was reduced by dipyridamole; 10^{-7} M dipyridamole reduced the ratio of tissue activity to activity in the medium (T/M ratio) from 8.09 ± 0.94 to 4.13 ± 0.59, while 10^{-6} and 10^{-5} M dipyridamole decreased the T/M ratio further to 1.79 ± 0.29 and 0.94 ± 0.12, resp. When a 10-ml sample of 10^{-4} M adenosine was incubated with 500 mg of ileal segment, about 50% of the adenosine in the medium disappeared in 5 min, with a slight concomitant recovery of the twitch response; 50% recovery occurred after 20 min. In the presence of 10^{-6} M dipyridamole, adenosine disappearance and recovery of the twitch response were much slower; 50% of the adenosine disappeared in 15 min, and 50% of the twitch response was restored in 60 min. It appears that dipyridamole potentiates the inhibitory actions of adenosine and adenine nucleotides on the ileal twitch response by inhibiting both the accumulation and the degradation of adenosine.

8650 ELECTRICAL ACTIVITY OF COLON OF THE NONSEDATED DOG. (Fre.) Bueno, L.; Garcia-Villar, R.; Ruckebusch, Y. (Ecole national veterinaire, 23, chemin des Capelles, 31076 Toulouse Cedex, France). *C. R Acad. Sci. [D] (Paris)* 285 (16):1463-1466; 1977.

Electromyographic studies of the colon were performed on nonsedated dogs that were fasted or that had ingested a standard meal. Electrodes were implanted in 10 anesthetized adult dogs in the wall of the ileum 2 cm above the ileo-colonic junction and in the wall of the colon, 5, 10, and 15 cm below the junction. One week after the electrodes were placed surgically, a second was inserted rectally to the ileocolonic junction to introduce endoluminal electrodes for comparative recordings in the nonsedated state. Both the intraparietal and endoluminal electrodes recorded two rhythms of colonic slow waves, with frequencies of 5.1 ± 1.3 cycles/min and 18.1 ± 1.1 cycles/min, resp. The electrograms produced by the endoluminal electrodes differed in two respects from the intraparietal recordings: (1) the amplitude of the rapid rhythms was in the neighborhood of 0.2 mV compared to 0.1 mV measured by the intraparietal electrodes and (2) the slower rhythm was recorded less than 2% of the time and was apparently superimposed on the rapid rhythm. Brief salvoes (1.4 ± 0.3 sec) of the faster rhythms appeared to be preferentially recorded by the endoluminal electrodes. It is concluded that the faster rhythm of the colonic slow waves arises in the muscularis mucosae, which is isolated from the muscular fibers of the wall by the submucosa in the dog.

8651 RELAXATIONS MEDIATED BY ADRENERGIC AND NONADRENERGIC NERVES IN HUMAN ISOLATED TAENIA COLI. (Eng.) Stockley, H. L.; Bennett, A. (Charing Cross Hosp. Medical Sch., London, W6 8RF, England). *J. Pharm. Pharmacol.* 29(9):533-537; 1977.

To investigate relaxations mediated by adrenergic and nonadrenergic nerves in isolated human taenia coli, macroscopically normal specimens of human ascending, transverse, and sigmoid colon, obtained at operation for colonic carcinoma or Crohn's disease, were studied. The strips of taenia responded to electrical stimulation at 4, 8, and 16 Hz with 0.3, 0.5, and 1 msec pulses, but most were unaffected at 0.1 msec. The order of magnitude of maximal relaxations induced by adrenergic blocking drugs was norepinephrine (0.2-1 µg/ml)>nicotine (1-40 µg/ml)≈ATP (90-1,440 µg/ml)>electrical stimulation. Electrical stimulation in the presence of hyoscine (2 µg/ml) caused relaxation followed by a rapid "after-contraction." Adrenergic blockade by oxprenolol (0.6-6 µg/ml) or guanethidine (20-30 µg/ml) caused relaxations to ATP to increase in three and decrease in one of five experiments and to nicotine at 16 Hz to decrease greatly or be wholly prevented. Relaxations to the effects of the indirectly-acting sympathomimetics tyramine (0.8-4 mg/ml, 0.5-5 min contact) and nicotine (4 µg/ml) were studied to examine interactions between adrenergic and nonadrenergic inhibitory nerves. Tyramine caused contraction followed by relaxation after washout, or only a delayed relaxation; the contractions were unaffected by tetrodotoxin (TTX, 1 µg/ml), dihydroergotoxine (1 µg/ml), or oxprenolol (5 µg/ml) and were enhanced by tranylcypromine (0.8-800 µg/ml). Relaxations to nicotine increased with contact time up to 4 min ($p<0.05$ for the difference in response between each time period) and were reduced by TTX to 1%, 2%, 6%, and 10% of controls with 0.5, 1, 2, and 4 min, resp., of contact time. Nicotine-induced relaxations that persisted in the presence of TTX seemed mainly adrenergic since oxprenolol (3 and 15 µg/ml) reduced them to 21% and completely prevented them, resp. Thus, adrenergic relaxations during stimulation occur only with short electrical pulses at high frequencies or with ganglion stimulants. The results with TTX and the sympathomimetics demonstrate that colonic adrenergic nerves are mainly preganglionic and possibly synapse with nonadrenergic inhibitory nerves. When the latter undergo direct maximal stimulation, responses to adrenergic nerve excitation are masked.

8652 A CIRCUMFERENTIALLY-SENSITIVE MINIATURE PRESSURE SENSOR FOR STUDY OF HUMAN ESOPHAGEAL MOTILITY. (Eng.) Kaye, M. D.; Showalter, J. P.; Rock, K. C.; Johnson, E. (Gastroenterology Unit, Univ. Vermont, Burlington, VT). *Med. Res. Eng.* 12(5):10-15; 1978.

8653 GASTRIC TRANSMURAL POTENTIAL DIFFERENCE IN RATS FED A RETINOL-FREE DIET. (Eng.) Sommer, H.; Kasper, H. (Medizinische Universitatsklinik, Luitpoldkrankenhaus, D-8700 Wurzburg, W. Germany). *Acta Hepatogastroenterol. (Stuttg.)* 25(2):136-138; 1978.

8654 SYNTHESIS AND BIOLOGICAL ACTIVITY OF 8-L-ALA-MOTILIN AND 8-D-ALA-MOTILIN. (Eng.) Shinagawa, S.; Fujino, M.; Yajima, H.; Segawa, T.; Okuma, Y. (Chemical Res. Lab., Takeda Chemical Industries, Ltd., Juso-honmachi, Yodogawa-ku, Osaka, 532, Japan). *Chem. Pharm. Bull.* 26(3): 880-884; 1978.

8655 INTRALUMINAL PRESSURE PROFILES AND MUCOSAL MOVEMENTS IN THE STOMACH AND DUODENUM. (Eng.) Keet, A. D.; Vermaak, J. C.; Mouton, J. (Dept. Radiology, Univ. Stellenbosch, Tygerberg 7505, South Africa). *Am. J. Gastroenterol.* 69(2):144-148; 1978.

8656 ANASTOMOSIS BY INVAGINATION: A TECHNIQUE TO PRESERVE ANAL SPHINCTER FUNCTION. (Eng.) Johnson, W. C. (Boston Veterans Admin. Hosp., Boston, MA 02130). *Dis. Colon Rectum* 21(3):188-195; 1978.

8657 THE LOGICAL ANALYSIS OF THE ELECTROENTEROGRAM. (Eng.) Wingate, D. L.; Barnett, T. (London Hosp. Medical Coll., Turner St., London E1 2AD, England). *Am. J. Dig. Dis.* 23(6):553-558; 1978.

8658 TESTS OF GASTROINTESTINAL MOTILITY. (Eng.) Connell, A. M. (Univ. Cincinnati Medical Center, Cincinnati, OH). *Clin. Gastroenterol.* 7(2):317-328; 1978.

See also, 8627, 8635, 8664, 8805, 8905.

SECRETION AND METABOLISM

8659 VARIATIONS IN PARASYMPATHETIC SECRETORY AND STRUCTURAL RESPONSES RESULTING FROM DIFFERENCES IN THE PRE-STIMULATION STATE OF PAROTID ACINI IN RATS: SOME FACTORS AFFECTING WATERY VACUOLATION. (Eng.) Garrett, J. R.; Thulin, A.; Kidd, A. (King's Coll. Hosp. Dental Sch., Denmark Hill, London SE5 8RX, England). *Cell Tissue Res.* 188(2):235-250; 1978.

8660 PROTEIN AND DNA LEVELS IN THE SUBMANDIBULAR SALIVARY GLANDS OF ISOPROTERENOL STIMULATED MICE. (Eng.) Feiglin, B.; Reade, P. C. (Dept. Dental Medicine and Surgery, Univ. Melbourne, Parkville, 3052, Victoria, Australia). *Aust. J. Exp. Biol. Med. Sci.* 56(Part 1):1-10; 1978.

8661 GASTROINTESTINAL HORMONES. (Eng.) McGuigan, J. E. (Univ. Florida Coll. Medicine, Gainesville, FL 32610). *Ann. Rev. Med.* 29:307-318; 1978.

8662 STUDIES ON KALLIKREIN IN THE DUCT SYSTEMS OF THE SALIVARY GLANDS OF THE CAT. (Eng.) Maranda, B.; Rodrigues, J. A. A.; Schachter, M.; Shnitka, T. K.; Weinberg, J. (Dept. Physiology, Univ. Alberta, Edmonton, Alberta T6G 2H7, Canada). *J. Physiol. (Lond.)* 276:321-328; 1978.

8663 EFFECTS OF A NEW SELECTIVE β_1-ADRENOCEPTOR AGONIST ON AMYLASE SECRETION FROM THE RAT PAROTID GLAND. (Eng.) Carlsoo, B.; Danielsson, A.; Henriksson, R. (Dept. Otolaryngology, Univ. Umea, Umea, S-901 87 Sweden). *Br. J. Pharmacol.* 62(3): 364-366; 1978.

See also, 8622, 8629, 8782.

SECRETION AND METABOLISM

Stomach

8664 ACID SECRETION AND MOTILITY OF ISOLATED MAMMALIAN GASTRIC MUCOSA AND ATTACHED MUSCULARIS EXTERNA. (Eng.) Yates, J. C.; Schofield, B.; Roth, S. H. (Div. Medical Physiology, Univ. Calgary, Calgary, Alberta T2N 1N4, Canada). *Am. J. Physiol.* 234(3):E319-E325; 1978.

A procedure for setting up a preparation of mammalian gastric mucosa from kitten or ferret is described, and the responses of both preparations to cholinergic, histamine, and pentagastrin stimulation were studied. The mucosa is stripped of its outer muscle coats except for a 2-mm strip of circular muscle fibers

running across the diameter of the preparation. The connections of this strip to the underlying mucosa are undisturbed. The preparation is immersed on the nutrient side in Krebs-Henseleit solution gassed with 95% O_2 and 5% CO_2 and on the secretory side in 5% dextrose, with both sides at 37 C. Acid secretion is determined by a pH stat method, and motility is recorded by a strain gauge attached to one end of the muscle strip. Secretory and motility responses are recorded for the three main parietal cell agonists, acetylcholine (ACh), pentagastrin, and histamine. Ferret membranes displayed a spontaneous rate of secretion of between 0.6 and 1.8 μEq H^+/cm^2 hr with a mode near 1 μEq/cm^2/hr, while the kitten membranes exhibited basal rates of acid secretion having a similar range of variation with a modal value near 0.7 μEq/cm^2/hr. The greatest secretory response to acetylcholine (ACh) stimulation in the ferret membranes was seen with a concentration of 10^{-4} M; a mean plateau of secretion of 4.4 μEq/cm^2/hr was established within 16 min. Smaller responses were seen with lower (10^{-5} M) and higher (10^{-3} M) ACh concentrations. Fundic smooth muscle response to ACh was rapid, and onset consisted of an abrupt large increase in the amplitude of spontaneous contractions, which declined shortly thereafter, and of a more sustained increase in basal tension. Although only a small increase in secretion was seen after an ACh dose as high as 10^{-3} M in the kitten fundic membranes, the kitten and ferret membranes were not greatly dissimilar in their sensitivity to cholinergic stimulation; however, the stable choline ester, urecholine, had to be used before the acid secretory response was fully manifested in the kitten preparation. The mean plateau acid secretory responses to 6.51×10^{-7} and 6.51×10^{-8} M pentagastrin in the ferret were 4.6 and 3.9 μEq/cm^2/hr, resp. The contractile response was similar to that produced by ACh. The kitten membrane was less sensitive to the hormone, both in terms of secretory and muscle responses. Acid secretion increased to 3 μEq/cm^2/hr after stimulation with 1.6×10^{-6} M histamine. The contractile response varied with the histamine dose. In the kitten, the secretory response to 1.6×10^{-6} M histamine was similar to that in the ferret; 1.6×10^{-5} M histamine produced a further increase, which was not seen in the ferret. Changes in contractile activity and basal tension were much more profound than those seen in the ferret. In both animals, the similarity between the motility responses produced by cholinergic stimulation and pentagastrin was notable.

8665 ULTRASTRUCTURAL CHANGES IN MOUSE PARIETAL CELLS AFTER HIGH H^+ SECRETION. (Eng.)
Ito, S.; Schofield, G. C. (Harvard Medical Sch., Boston, MA). *Acta Physiol. Scand.* (Special Suppl.): 25-34; 1978.

Further elucidation of parietal cell ultrastructural changes found during different secretory stages, particularly when acid secretion was stopped, were studied. Perfused stomachs of anesthetized mice were monitored for acid secretion and then fixed at known periods of secretory activity and examined ultrastructurally. The characteristic morphology of active and of nonsecreting parietal cells was confirmed and some intermediate stages were observed. In histamine- and carbachol-stimulated parietal cells, taken from mice secreting as much as 1 μEq/g body weight/15 min at the peak period of secretion, the tubulovesicular compartment was greatly or completely depleted, and there was a great increase in the number and length of microvilli, which tended to occlude the luminal space of the canaliculi. In contrast to previous observations, this study suggests that the lumen of the gastric gland and the intracellular canaliculi are separate compartments. Observations in cat parietal cells, which showed an intracellular canaliculus with an open lumen, a canaliculus containing a section of a gastric spirillum, and lysosomal bodies containing spirilla, supported the internalization of the intracellular canaliculi. In mouse parietal cells recovering from high secretory activity, the lumen of the canaliculus appeared distended. Although elements of the tubulovesicular system came into close approximation with the plasma membrane, there was no evidence that there was any direct continuity between these two membrane compartments. Numerous small multivesicular bodies as well as lysosomes were apparent. When the parietal cells were returning to an inactive state, the microvilli seemed to lose some of their rigidity and were often pleomorphic or bulbous in shape. In some of these microvilli, tubular membranes were invaginated into the microvillar core. Concentric membrane profiles were present in the cytoplasm of recovering parietal cells. Associated with these concentric profiles were flattened vesicles and coated vesicles, the latter of which were either joined to the plasma membrane or free in the cytoplasm. In some cells, the cytoplasm contained clear, rounded or elongated bodies surrounded by two layers of closely apposed membranes. It is suggested that these morphological features and other processes that may not be apparent by current tissue preparation techniques are involved in the movement of membrane to and from the tubulovesicular compartment and the plasma membrane.

8666 THE ROLE OF ASPIRIN IN GASTRIC ULCERATION: SOME FACTORS INVOLVED IN THE DEVELOPMENT OF GASTRIC MUCOSAL DAMAGE INDUCED BY ASPIRIN IN RATS EXPOSED TO VARIOUS STRESS CONDITIONS. (Eng.)
Rainsford, K. D. (Univ. Tasmania Medical Sch., G.P.O. Box 252C Hobart, Tasmania, Australia). *Am. J. Dig. Dis.* 23(6):521-530; 1978.

The development of gastric mucosal damage induced by aspirin was studied in hooded rats stressed by physical (cold) and disease (adjuvant-induced arthritis, acute pain, inflammation) conditions to establish whether these stress states increase the susceptibility of gastric mucosa to aspirin damage. To elucidate the mechanisms involved in the physical stress plus aspirin interaction, a variety of pharmacological agents was also studied. Gastric irritation was measured 2 hr after the p.o. administration of aspirin (100-250 mg/kg) in a single dose or once daily doses for up to 10 days. Physical stress increased the sensitivity of the gastric mucosa to aspirin damage (there were 2.7 ± 1.0 lesions found in male rats treated with as-

pirin alone and 24.8 ± 9.5 lesions found in such rats with aspirin plus cold-stress treatment $p<0.05$). No similar increases were noted in disease-stressed animals. Pharmacological inhibitors/antagonists of stress-mediated stomach ulceration including atropine methyl nitrate (5-10 mg/kg, p.o.), bilateral vagotomy, phentolamine (5 mg/kg, i.p.), propranolol (5 mg/kg, i.p.), metyrapone (250 µg/kg, i.m. for 4 days), metiamide (3 mg/kg, i.p.), cimetidine (5-40 mg/kg, i.p. or 40 mg/kg, p.o.), prostaglandins (PG) E_1 (2 mg/kg, i.p.), PGE_2 (5 mg/kg, i.p.), and 15(R),15-methyl PGE_2 (50 µg/kg, i.p.) were studied. Use of the anticholinergic agent, atropine methyl nitrate (ED_{50}, 5 mg/kg following 200 mg/kg aspirin to stressed rats), bilateral vagotomy, or combined treatment with α- and β-adrenergic blockers were effective in reducing the gastric mucosal irritation induced by aspirin plus cold stress. The antisecretory agents prevented the effects of aspirin plus cold stress, prostaglandins allowing no lesion formation at all. Levels of corticosterone in the blood and the distribution and metabolism of ^{14}C-labeled aspirin in stressed and nonstressed rats were measured; as no differences in mucosal uptake of ^{14}C-aspirin were observed, accelerated uptake of the drug was eliminated as a factor in the development of gastric ulceration. The results show a major involvement of the parasympathetic-vagal, sympathetic, and histamine-producing systems, but not the adrenocortical axis, in the development of gastric mucosal damage induced by aspirin plus physical stress.

8667 THE EFFECT OF BETA-ADRENERGIC AND CHOLINERGIC BLOCKADE ON THE CIRCADIAN RHYTHM OF GASTRINS IN SERUM. (Eng.) Christensen, K. C.; Stadil, F.; Malmstrom, J.; Rehfeld, J. F. (Rigshospitalet, 2100 Copenhagen, Denmark). *Scand. J. Gastroenterol.* 13(3):263-272; 1978.

Studies were carried out in 15 healthy subjects to determine whether different gastrin components in the serum show circadian rhythms, and if so, whether beta-adrenergic and cholinergic blockade affect the serum concentration of gastrins. The subjects were investigated during p.o. treatment with the beta-adrenergic blocking agent propranolol (20 mg), the anticholinergic drug glycopyrron (1 mg), both drugs, or neither drug. Gastrin concentrations were measured with an antiserum that measures all four main gastrin components in serum, and with a gastrin-17-specific antiserum. A circadian rhythm was observed with both antisera. The total serum immunoreactivity increased from 39 ± 3 pg/ml at 0800 hr to a peak of 77 ± 7 pg/ml at 2300 hr, followed by a nadir of 38 ± 2 pg/ml at 0400 hr. The circadian rhythm was maintained during administration of both drugs, but the concentrations of component III (gastrin-17) were reduced by beta-adrenergic blockade (from 733 ± 71 to 599 ± 28 pg/ml/hr, $p=0.05$), while treatment with glycopyrron increased the concentrations of the other gastrin components (from 490 ± 63 to 647 ± 57 pg/ml/hr, $p=0.01$). The inhibition of gastric acid secretion by anticholinergics is presumably due to an action on the parietal cells; according to this study, the inhibition is connected by an increased gastrin stimulus. Beta-adrenergic blockade, however, seems to reduce the gastrin stimulus and might be of therapeutic interest in duodenal ulcer disease.

8668 THE INFLUENCE OF ATROPINE ON INSULIN- AND 2-DEOXY-D-GLUCOSE (2-DG)-INDUCED GASTRIN RELEASE IN DOGS. (Eng.) Ornsholt, J.; Brandsborg, O.; Brandsborg, M.; Lovgreen, N. A.; Amdrup, E. (Kommunehospitalet, DK-8000 Arhus C, Denmark). *J. Surg. Res.* 23(4):246-250; 1977.

To evaluate whether parietal cells and gastrin cells differ in sensitivity to atropine, the influence of this drug on insulin- and 2-deoxy-D-glucose (2-DG)-induced gastric acid secretion and gastrin release was studied in gastric fistula dogs. 2-DG (200 mg/kg, i.v.) caused a pronounced rise in acid secretion within the first 15 min after injection ($p<0.05$), as compared with the basal level. Insulin (0.25 U/kg, i.v.) produced a marked increase in acid secretion over the basal level, with a peak output in the 30- to 45-min period. Insulin also produced increases in the serum gastrin level, which were significantly elevated above the basal level from 30 to 90 min after injection ($p<0.05$). The mean gastrin concentration stimulated by 2-DG showed an immediate rise to a peak value 15 min after the injection ($p<0.01$). Then a marked decrease occurred, reaching basal concentration at 45 min. Atropine pretreatment (0.05 mg/kg, s.c.) caused an almost complete block of insulin-induced acid secretion during the first three 15-min periods, but a weak secretion was recorded about 60 min after the injection, followed by a gradual decrease to the basal level at 120 min. Atropine initially blocked 2-DG-stimulated acid output, but thereafter a constant increase in acid output was observed. The acid output between 105 and 135 min was significantly higher than the basal secretion ($p<0.05$). Atropine in doses of 0.05 to 0.2 mg/kg had no effect on 2-DG- or insulin-stimulated gastrin release. Gastrin release during hypoglycemia in the dog might be cholinergic but resistant to atropine doses that are sufficient to block acid secretion.

8669 THE INHIBITION OF GASTRIN PRODUCTION BY CALCITONIN IN THE AGED. (Ita.) Passeri, M.; Palummeri, E.; Soncini, G.; Monica, C.; Ferretti, P. G.; Galvagni, R. (Cattedra di Gerontologia e Geriatria dell'Universita degli Studi di Parma, Parma, Italy). *G. Clin. Med.* 58(9/10):321-338; 1977.

The effect of calcitonin on gastrin production in the aged was studied. A group of volunteers (average age 72 ± 3 yr) with no apparent gastrointestinal disorders was given pig calcitonin or salmon calcitonin (24 mg/kg or 20 µg, resp., i.v.) prior to a high-protein (9.5 g/250 ml) meal. The same patients served as controls when they received only the high-protein meal in a test carried out 72 hr prior to the calcitonin experiment. Blood samples

taken during both studies were analyzed for calcium, gastrin, and insulin levels. In the 16 patients treated with pig calcitonin, the gastrin response peaked at 15 min after the meal at 130% of its basal value, whereas it reached 190% of its basal value at this time when calcitonin was not given first. At 90 min after the meal, the calcium level was approximately 9.5 mg/100 ml in the controls but about 9.2 mg/100 ml in the treated group. The insulin level was approximately 12 μU/ml 30 min after the meal in the controls but about 8 μU/ml when in the treated group. For those treated with salmon calcitonin, the gastrin values reached 160% of base value in the controls but only about 110% of base value in the treated group 20 min after the meal. The calcium level 10 min after the meal was approximately 10.1 mg/100 ml in the controls and about 9.8 mg/100 ml in the treated group. The insulin level was about 19 μU/ml 20 min after the meal in the controls but about 7 μU/ml in the treated group. The authors state that the results were statistically significant between the control and treated group for both pig and salmon calcitonin 20 and 30 min after the meal. It is suggested these effects might be important in postprandial regulation of calcemia, in the nourishment of bone, and in senile osteoporosis.

8670 INHIBITION BY SOMATOSTATIN (GROWTH-HORMONE RELEASE-INHIBITING HORMONE, GH-RIH) OF GASTRIC ACID AND PEPSIN AND G-CELL RELEASE OF GASTRIN. (Eng.) Barros D'Sa, A. A. J.; Bloom, S. R.; Baron, J. H. (Singleton Hosp., Swansea, Wales). *Gut* 19(4):315-320; 1978.

The effects of somatostatin (cyclic growth hormone release-inhibiting hormone, GH-RIH) on gastric acid, gastrin, and pepsin secretion in response to insulin hypoglycemia, a meat meal histamine, and pentagastrin were studied in dogs with gastric fistulae. During experiments in which secretion was stimulated with histamine and pentagastrin, somatostatin was infused in the second hour of a 3-hr infusion of submaximal doses of histamine acid phosphate (0.5 μg/kg/min) and pentagastrin (1 μg/kg/hr). In the insulin experiments, insulin (0.2 U/kg) was given as a single i.v. injection, and somatostatin (5 or 8 μg/kg/hr) was infused for 30 min before and at 90-120 min after the insulin injection. During meal-stimulated experiments, somatostatin was given i.v. in the first 2 hr of a 3-hr experiment. In insulin-stimulated secretion experiments, the mean gastrin increment was inhibited by 70% after the lower dose and by 86% after the higher dose of somatostatin, almost identical with the inhibition of insulin-stimulated acid output of 74% and 85%, resp., after these doses. After a meal, inhibition of acid output was greater with increasing doses of somatostatin. At the lowest somatostatin dose (0.5 μg/kg/hr), there was a rebound in secretion after the somatostatin infusion was stopped, with the acid output exceeding control levels. During the 2-hr infusion with 1 μg/kg/hr somatostatin, gastric acid secretion was totally inhibited in every dog, with some return of secretion after the infusion was terminated. Acid secretion was completely inhibited throughout the 3 hr with the other four doses (2, 4, 8, and 16 μg/kg/hr). Meal-stimulated gastrin inhibition was also dose-dependent. Pepsin outputs after somatostatin were inhibited less than were acid outputs. Histamine-stimulated acid secretion was inhibited by somatostatin, and pentagastrin-stimulated acid output decreased immediately and rapidly after somatostatin was infused. Acid and pepsin outputs stimulated by pentagastrin were reduced by 94% and 76%, resp., after somatostatin. Basal plasma gastrin levels were not affected by histamine or pentagastrin infusions. During somatostatin infusion, the basal plasma levels were depressed, with some recovery after somatostatin infusions were stopped. These studies show that somatostatin inhibits the release of acid, pepsin, and gastrin from parietal, chief, and G-cells in response to a meal; of acid and gastrin in response to insulin; and of acid and pepsin in response to pentagastrin and histamine.

8671 INHIBITION OF GASTRIC ACID SECRETION IN DOGS BY A NEW SYMPATHOMIMETIC DRUG. (Eng.) Gottrup, F.; Ornsholt, J. (Dept. Anatomy, Univ. Arhus, 8000 Arhus C, Denmark). *Scand. J. Gastroenterol.* 13(3):337-343; 1978.

The inhibitory effect of a new β_2-agonist, salmefamol, on pentagastrin-induced acid secretion was studied in conscious gastric-fistula dogs. Maximal pentagastrin-induced acid secretion was significantly reduced at i.v. doses of 0.1, 0.2, 0.4, and 0.8 μg/kg/min salmefamol. The inhibition was 43 ± 8% for the lowest dose (0.05 μg/kg/min) and 89 ± 4% for the highest dose (0.8 μg/kg/min). Inhibition occurred within 15 and 30 min, and the effect was maximal after 45 min. A dose-related decrease in the titratable acidity and volume was demonstrated. The maximal decrease was 34 ± 3% ($p<0.05$) and 83 ± 5% ($p<0.05$), resp. Salmefamol caused an increase in the pulse rate of 34 ± 5% (at the lowest dose) to 96 ± 22% (at the highest dose). Propranolol (0.2 mg/kg, i.v.) prevented the inhibition of acid output as well as the increase in pulse rate. Practolol (0.4 and 1.0 mg/kg, i.v.) had no effect on the inhibition of acid secretion but prevented the increase in pulse rate. Dose-response experiments with 0.25-8.00 μg/kg/hr pentagastrin and salmefamol at 0.1 μg/kg/min showed a decrease in the calculated maximal response and an unchanged half maximal acid response. It is concluded that salmefamol strongly inhibits pentagastrin-induced acid output in the dog, and that the inhibition follows noncompetitive kinetics. The mechanism of gastric secretion probably involves a β_2-receptor.

8672 BACTERIAL FLORA AND NITRITE PRODUCTION IN THE STOMACH AFTER GASTROENTEROSTOMY. EXPERIMENTAL ASPECTS OF THE PATHOGENESIS OF CARCINOMA IN THE OPERATED STOMACH. (Ger.) Schlag, P.; Wonka, W.; Meyer, H.; Feyerabend, G.; Merkle, P. (Abteilung fur Allgemeine Chirurgie des Departments fur Chirurgie, Universitat Ulm, Steinhovelstr. 9, D-7900

Ulm, W. Germany). *Langenbecks Arch. Chir.* 344(2): 109-114; 1977.

The effect of gastroenterostomy without entero-anastomosis, of gastroenterostomy with Roux-en-Y anastomosis, and of gastrotomy on the nitrite-decomposing bacteria of the stomach was studied in specific pathogen-free male Wistar rats to investigate the increased gastric cancer risk following gastric surgery. Gastroenterostomy without enteroanastomosis caused a marked increase in the proportion of the nitrite-decomposing bacteria (*Escherichia coli, Lactobacillus acidophilus, Peptostreptococcus productus,* and *Eubacterium contortum*), and, accordingly, a sixfold increase in the nitrite concentration in the gastric juice 35 weeks after operation. No significant change was seen in the proportion of the nitrite-decomposing bacteria or in the nitrite level of the gastric juice of the rats that underwent gastroenterostomy with Roux-en-Y anastomosis compared with the gastrotomied (control) rats. Increased nitrite concentration in the gastric juice may lead to an enhanced production of carcinogenic nitrosamines.

8673 EFFECT OF BILE ON LYSOSOMAL STABILITY IN THE MUCOSA OF THE CANINE GASTRIC ANTRUM. (Eng.) Waldron-Edward, D.; Boutros, M. I. R.; Himal, H. S. (Dept. Experimental Surgery, McGill Univ., 740 McGregor, Montreal, Quebec H3A 1A4, Canada). *Gastroenterology* 73(5):980-984; 1977.

The activities of bound and free hydrolases and the response of the membranes to hypoosmotic (distilled water, 4 C, 240 min) and mechanical (tissue grinder) stress were studied in explanted canine antral mucosa to investigate the role of bile reflux in ulcerogenesis. Tissue and bile were obtained from the same mongrel dogs and the explanted mucosa was treated with bile or bile and then acid and fractionated to obtain the lysosome-rich pellet. The particulate enzymes, acid phosphatase (AcP), cathepsin D (CD), β-galactosidase (βGAL), α-galactosidase (α-GAL), β-*N*-galactosaminidase (β-*N*-GAL), β-*N*-glucosaminidase (β-*N*-GLU), and α-fucosidase (α-FUC), varied considerably in the stability of their binding to subcellular organelles when the tissue had been treated with acid (120 mEq HCl)-saline (40 mEq NaCl) for 60 min: α-FUC and β-*N*-GAL activities remained tightly bound after hypoosmotic shock or mechanical stress, whereas about three times as much β-GAL and β-*N*-GLU activities (percentage of initially bound activity) were released. When tissue was treated with bile for 60 min, very little enzyme activity was lost from membranes during subsequent treatments; however, hypoosmotic shock doubled the release of CD into the soluble fraction ($p<0.001$) compared with nontreated mucosa subjected only to acid-saline. Similarly, soluble protein increased by 75% ($p<0.001$). The pellets from tissue treated first with bile (30 min) and then acid-saline (15 or 30 min) were quite fragile to hypoosmotic shock, the fragility increasing with time of exposure to acid-saline; the percentage of previously bound activity lost was significant in every case after the 30-min acid-saline treatment ($p<0.01$ for β-*N*-GLU and $p<0.001$ for AcP, CD, β-GAL, α-GAL, β-*N*-GAL, and α-FUC). However, this tissue yielded pellets that were markedly resistant to mechanical stress and only AcP, β-*N*-GLU, β-*N*-GAL, and α-FUC activities were released in significant ($p<0.001$) amounts. The total free plus labilizable activity of most of the acid hydrolases of the mucin-cleared, nuclei-free homogenates from the insulted mucosa decreased slightly from control levels, with a marked increase in protein level and decrease in enzyme specific activity in this fraction. The increased levels of free enzyme appeared in the supernatant fraction, reaching a significant level ($p<0.01$ or 0.05) in the bile-then acid-treated samples. It is concluded that, while bile alone has little direct effect on the membrane binding or activation of most acid hydrolases, it facilitates the influx of H^+, thus releasing the acid hydrolases into an active acidic environment and exposing the entire mucosal cell structure to abnormal biodegradation or autolysis.

8674 16,16-DIMETHYL PROSTAGLANDIN E_2 IS NOT A BARRIER BREAKER. (Eng.) Bolton, J. P.; Cohen, M. M. (Univ. British Columbia Faculty Medicine, Vancouver, British Columbia, Canada). *Surg. Forum* 28:363-366; 1977.

8675 MUCOSAL EROSIONS AND DEPRESSED GASTRIC PROTEIN SYNTHESIS IN THE SEMISTARVED RAT. (Eng.) Hargrove, W. C.; Mullen, J. L.; Stein, T. P.; Oram-Smith, J. C.; Rosato, E. F. (Univ. Pennsylvania Sch. Medicine, Philadelphia, PA). *Surg. Forum* 28:366-368; 1977.

8676 EVIDENCE FOR SECRETION OF A CALCIUM-LOWERING FACTOR FROM THE STOMACH. (Eng.) Shen, K. L.; McCauley, R.; Hill, B.; Kaplan, E. L. (Univ. Chicago Pritzker Sch. Medicine, Chicago, IL). *Surg. Forum* 28:392-394; 1977.

8677 CENTRAL AND PERIPHERAL ADRENERGIC MECHANISMS REGULATING GASTRIC SECRETION IN THE RAT. (Eng.) Yamaguchi, I.; Hiroi, J.; Kumada, S. (Fujisawa Pharmaceutical Co. Ltd., 1-6, 2-Chome, Kashima, Yodogawa-ku, Osaka 532, Japan). *J. Pharmacol. Exp. Ther.* 203(1):125-131; 1977.

8678 EFFECTS OF VARIOUS TYPES OF VAGOTOMY ON GASTRIN RELEASE AND GASTRIC SECRETION IN DOGS. (Eng.) Shiratori, T.; Okabayashi, T.; Nishigori, M.; Sakuro, R.; Shimano, Y. (Nara Medical Univ., Kashihara 634, Japan). *Tohoku J. Exp. Med.* 122(3):209-222; 1977.

8679 THE TURNOVER OF MUCIN GLYCOPROTEIN IN THE STOMACH. (Eng.) Waldron-Edward, D. (McGill Univ., Donner Building, P.O. Box 6070, Station A, Montreal, Quebec, Canada). *Adv. Exp. Med. Biol.* 89:301-308; 1977.

8680 STRUCTURE AND FUNCTION IN GASTRIC MUCUS. (Eng.) Allen, A. (Dept. Physiology, Univ. Newcastle upon Tyne, Newcastle upon Tyne, NE1 7RU, England). *Adv. Exp. Med. Biol.* 89:283-299; 1977.

8681 EFFECT OF PENTAGASTRIN ON CHOLINE ACETYLTRANSFERASE AND ACETYLCHOLINESTERASE ACTIVITIES IN VARIOUS TISSUES OF THE DIGESTIVE TRACT OF RATS. (Eng.) Majumdar, A. P. N.; Nakhla, A. M. (Inst. Medical Biochemistry, Univ. Aarhus, DK-8000 Aarhus C, Denmark). *Biochem. Biophys. Res. Commun.* 81(4):1241-1247; 1978.

8682 THE NATURE OF ABH BLOOD GROUP ANTIGENS IN HUMAN GASTRIC SECRETION. (Eng.) Slomiany, A.; Slomiany, B. L.; Glass, G. B. J. (New York Medical Coll., New York, NY 10029). *Biochim. Biophys. Acta* 540(2):278-284; 1978.

8683 BIOLOGICAL PROPERTIES OF THE GASTRIC JUICE INHIBITORY SUBSTANCE PRODUCED BY Streptomyces bottropensis F4708. (Eng.) Mimura, T.; Muto, N.; Oda, Y.; Tanaka, N.; Aonuma, S. (Faculty Pharmaceutical Sciences, Osaka Univ., 133-1, Yamadakami, Suita-shi, Osaka 565, Japan). *Chem. Pharm. Bull.* 26(3):998-1002; 1978.

8684 CHANGES IN ELECTROENCEPHALOGRAMS OF RATS DURING THE DEVELOPMENT OF STRESS ULCER. (Eng.) Harada, M.; Ishii, T.; Yano, S. (Faculty Pharmaceutical Sciences, Univ. Chiba, Yayoi-cho, Chiba 280, Japan). *Chem. Pharm. Bull.* 26(3):746-751; 1978.

8685 ZINC CONCENTRATION IN THE PLASMA AND STOMACHS OF RATS WITH GASTRIC ULCERATION. (Eng.) Jaffarian, S.; Yazdani, C.; Djahanguiri, B. (Sch. Medicine, Univ. Tehran, Tehran, Iran). *Iar. J. Med. Sci.* 14(4):485-486; 1978.

8686 ACID SECRETION AND CYCLIC NUCLEOTIDE CONTENT OF THE GUINEA PIG ISOLATED STOMACH IN THE PRESENCE OF THE IONOPHORE A23187. (Eng.) Canfield, S. P.; Curwain, B. P.; Spencer, J. (St. Mary's Hosp. Medical Sch., London W2, England). *Acta Hepatogastroenterol. (Stuttg.)* 25(2):155-157; 1978.

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8688 A CORRELATIVE STUDY OF THE ANTIULCER EFFECTS OF ZINC SULPHATE IN STRESSED RATS. (Eng.) Cho, C. H.; Ogle, C. W. (Faculty Medicine, Univ. Hong Kong, Hong Kong). *Eur. J. Pharmacol.* 48(1):97-105; 1978.

8689 EFFECTS OF DRUGS INTERFERING WITH CATECHOLAMINE METABOLISM ON STRESS-INDUCED GASTRIC ULCERS IN THE RAT. (Eng.) Hano, J.; Bugajski, J.; Danek, L. (Inst. Pharmacology, Polish Acad. Sciences, 31-344 Cracow, Poland). *Pol. J. Pharmacol. Pharm.* 29(6):629-638; 1977.

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8691 DEVELOPMENT AND APPLICATION OF H-2 RECEPTOR ANTAGONISTS [Abstract]. (Eng.) Parsons, M. E. (Smith, Kline and French Lab., Welwyn Garden City, England). *Scott. Med. J.* 22(4):292-293; 1977.

8692 DO β ADRENERGIC AGENTS DIRECTLY STIMULATE GASTRIN SECRETION? (Eng.) Kondo, T.; Magee, D. F. (Creighton Univ. Sch. Medicine, 2500 California St., Omaha NB 68178). *Experientia* 34(5):607; 1978.

8693 EFFECTS OF PROSTAGLANDIN E_2 ON GASTRIC ACID SECRETORY FUNCTION AND ULCEROGENESIS INDUCED BY DIFFERENT METHODS (EXPERIMENTAL STUDY). (Eng.) Vasilenko, V. H.; Grebenev, A. L.; Kochina, E. N.; Grebeneva, L. S.; Poloschenkova, L. A.; Tsvetkova, L. I. (First Moscow Medical Sechenov Inst., Moscow, USSR). *Gastroenterol. Jpn.* 13(1):1-7; 1978.

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8695 H^+ TRANSPORT IN GASTRIC MICROSOMES: TOWARDS A MOLECULAR MODEL FOR SECRETION. (Eng.) Michelangeli, F.; Proverbio, F. (Centro de Biofisica y Bioquimica, Instituto Venezolano de Investigaciones Cientificas, Caracas, Venezuela). *Acta Physiol. Scand.* (Special Suppl.):399-407; 1978.

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8697 HEMODYNAMIC ASPECTS OF STRESS-INDUCED GASTRIC MUCOSAL LESIONS IN PIGLETS. (Eng.) Hottenrott, C.; Seufert, R.; Becker, H.; Junghanns, K. (Chirurgische Universitats-Klinik, 6900 Heidelberg, W. Germany). *Chir. Gastroenterol.* 11(3):303-306; 1977.

8698 HAEMOSTATIC EFFECT OF TRIGLYCYLVASOPRESSIN ON BLEEDING FROM GASTRIC MUCOSAL LESIONS IN THE RAT. (Eng.) Wetterlin, S.; Ahlgren, I.; Aronsen, K. F.; Mulder, J. F. (Malmo General Hosp., Malmo, Sweden). *Res. Exp. Med. (Berl.)* 172(2): 131-135; 1978.

See also, 8623, 8653, 8778, 8844, 8847, 8850, 8876, 8878, 8896, 8905, 8910, 8914, 8917, 8918, 8923, 9149.

SECRETION AND METABOLISM

Pancreas

8699 COMPARISON OF TWO METHODS FOR DETERMINING THE DOSE-EFFECT RELATIONSHIPS BETWEEN CHOLECYSTOKININ AND EXOCRINE PANCREATIC FUNCTION IN THE DOG. (Ger.) Singer, M. V.; Dermol, P.; Mendes de Oliveira, J. P.; Sarles, H. (Unite de Recherches de Pathologie Digestive, INSERM, U 31, 46, chemin de la Gaye, F-13009 Marseille, France). *Z. Gastroenterol.* 16(2):95-100; 1978.

Dose-response curves to cholecystokinin (CCK) for pancreatic flow rate, bicarbonate output, and protein output were determined by two different techniques in three conscious dogs with gastric and duodenal Thomas fistulas. With the continuous technique (CT), the CCK dose was doubled each 45 min (3, 6, 12, and 24 Crick-Harper-Raper U/kg/hr). With the single-dose technique (SDT), only one of the above doses of CCK was administered on each test day. CCK was administered together with secretin (1 U/kg/hr, i.v.). The dose-response curves for pancreatic flow and bicarbonate output did not differ with the two techniques. At low CCK doses (3, 6, or 12 U/kg/hr), there was no difference between the two methods concerning the protein output. At the highest CCK dose (24 U/kg/hr), the maximum protein output was reached using the SDT; this output was significantly higher than that obtained with the CT ($p<0.01$). Consequently, the SDT is the method of choice for the determination of the maximum protein output, while the CT is sufficient for the determination of dose-effect relationships between submaximal CCK doses and protein output.

8700 EFFECTS OF HYPERTONIC GLUCOSE AND AMINO ACID INFUSIONS ON PANCREATIC EXOCRINE FUNCTION. (Eng.) Saito, Y.; Tokutake, K.; Matsuno, S.; Noto, N.; Honda, T.; Sato, T. (Tohoku Univ. Sch. Medicine, Sendai 980, Japan). *Tohoku J. Exp. Med.* 124(2):99-115; 1978.

The inhibiting effects of an i.v. infusion of 20% glucose, 30% glucose, 12% amino acids, or 20% mannitol (200 ml/hr for 1 hr or more) on pancreatic exocrine function were studied in mongrel dogs with chronic gastric and pancreatic fistulae. The effects of glucagon injected i.v. at intervals of 50 min in increasing doses of 2, 8, 16, and 32 µg/kg were measured (glucagon loading test). Basal stimulation by secretin and pancreozymin (continuous i.v. infusion of 0.025 U/kg/min in conscious dogs and 0.05 U/kg/min in anesthetized dogs) was also measured and showed a plateau in the volume of secreted pancreatic juice at 50-60 min after the start of infusion. Satisfactory results were obtained only with conscious dogs except in the measurement of immunoreactive glucagon (IRG). Infusion with 20% glucose, 30% glucose, or 12% amino acids for 1 hr significantly decreased both volume of pancreatic juice ($p<0.001$, $p<0.01$, $p<0.001$, resp.) and amylase output ($p<0.01$ for all 3 solutions) compared with preloading levels. In addition, both 30% glucose and 12% amino acids were more potent inhibitors than was 20% glucose ($p<0.05$ for both solutions). No changes in bicarbonate concentration were noted. The infusion of 20% mannitol for 2 hr produced an initial drop in the volume of pancreatic juice and in amylase output compared with preloading levels (both decreases were maximal at 10 min, $p<0.02$ and $p<0.05$, resp.), but at 50 min, both levels reverted to initial levels despite continuing infusion. When the 12% amino acid infusion was continued for 2 hr, however, both levels remained suppressed throughout the infusion. These differences in pancreatic response to 20% mannitol and to solutions having osmotic pressures equal to or greater than 20% mannitol showed that osmotic pressure does not exert a significant influence on the inhibitory effects of the test solutions. The 20% and 30% glucose infusions elevated blood sugar levels to average values of 261 and 367 mg/100 ml, resp. Increases in the levels of immunoreactive insulin (IRI) were produced by both glucose infusions and by the 12% amino acid infusion (levels never exceeded 73 µl/ml). There was an increase in the level of IRG following the 12% amino acid infusion but no trend of increase following either glucose infusion. Infused glucagon produced transitory inhibition of pancreatic juice secretion and amylase excretion, with the reduced values tending to return to initial levels 30 min after each injection. The greatest inhibition of pancreatic exocrine secretion in these experiments was produced by the amino acid infusion; IRI and IRG levels rose markedly. The stimulated glucagon release contributed to the suppression of pancreatic secretion. The infusion rate used in these studies was several times greater than the rate used clinically for hyperalimentation in adults with acute pancreatitis,

but the solution concentrations are those used routinely. The marked suppression of pancreatic secretion observed tends to support the hypothesis that hyperalimentation with hypertonic glucose and amino acids is of value in the treatment of acute pancreatitis.

8701 THE EFFECT OF PROLACTIN ON CANINE PANCREATIC SECRETION: IMPLICATIONS ON THE PATHOGENESIS OF THE PANCREATITIS OF PREGNANCY. (Eng.) Rosenberg, V.; Dreiling, D. A. (Mt. Sinai Sch. Medicine, City Univ. New York, 100th St. and Fifth Ave., New York, NY 10029). *Am. J. Gastroenterol.* 67(4):354-361; 1977.

The effect of the acute administration of prolactin on canine pancreatic secretion was studied to investigate the possibility that the pancreatitis of pregnancy may have a hormonal etiology. Pancreatic secretion was studied in 10-min samplings for 1 hr after the continuous i.v. pump infusion of secretin (0.3 U/kg/min) plus cholecystokinin-pancreozymin (CCK-PZ, 0.05 U/kg/min). The study was continued for an additional 3 hr after bolus administration of 2 mg prolactin. The average hourly volume, bicarbonate, and protein outputs during infusion of secretin and CCK-PZ were 104.5 ml, 13.8 mEq, and 1,298 mg, resp. The average hourly concentrations of bicarbonate and protein during this time were 132 mEq/l and 12.4 mg/ml. The average values during the first hour after prolactin injection were 84.7 ml, 9.3 mEq, and 1,088 mg for volume, bicarbonate, and protein output, resp., and 109 mEq/l and 12.8 mg/ml for bicarbonate and protein concentration, resp. During the second hour, these levels were 100.4 ml, 10.1 mEq, 799 mg, 101 mEq/l, and 7.9 mg/ml, resp. At hour 3, the levels were 101.6 ml, 9.7 mEq, 790 mg, 95 mEq/l, and 7.7 mg/ml, resp. These results show that prolactin diminishes the pancreatic response to continuous secretin plus CCK-PZ infusion. Protein enzyme concentration was unaltered, so that protein output was maintained. These alterations produce a low-flow enzyme-concentrated juice that would afford the possibility of small duct blockade by protein plug, thus explaining the pancreatitis of pregnancy.

8702 EFFECT OF SOMATOSTATIN ON BASAL AND STIMULATED PANCREATIC SECRETION IN THE RAT. (Eng.) Folsch, U. R.; Lankisch, P. G.; Creutzfeldt, W. (Dept. Medicine, Univ. Gottingen, Gottingen, W. Germany). *Digestion* 17(3):194-203; 1978.

The response to somatostatin of basal and cholecystokinin (CCK)- and secretin-stimulated pancreatic secretion was studied in anesthetized male Wistar rats. The i.v. infusion or bolus injection of somatostatin was always preceded by at least 30 min of 0.15 M NaCl infusion. Dose schedules were (1) somatostatin, 100 µg/hr by infusion or a bolus injection of 50 µg/100 g, (2) 3 Ivy dog U (IU) CCK-PZ/100 g/hr alone or combined with 50 or 100 µg/100 g/hr somatostatin, and (3) 0.8 clinical U (CU) secretin/100 g/hr for 60 min followed by the same dose of secretin either alone or combined with 50, 100, or 200 µg/100 g/hr somatostatin. The basal secretion of amylase was markedly inhibited by the 100 µg/100 g/hr somatostatin dose; however, the bolus injection of 50 µg/100 g somatostatin produced a fourfold increase in amylase secretion for 10-20 min followed by a return to the basal level. No changes in volume of secretion were observed. The combination of 3 IU/100 g/hr of CCK with 100 µg/100 g/hr somatostatin caused strong inhibition of amylase and trypsin secretion. The enzyme secretion increased immediately after termination of the somatostatin infusion, reflecting the short (3 min) half-life of CCK and demonstrating the reversibility of the inhibitory action of somatostatin. The CCK-stimulated volume output was markedly reduced by an infusion of 100 µg/100 g/hr somatostatin. The inhibition continued after the termination of the somatostatin infusion. No significant changes in the pattern or magnitude of secretin-stimulated pancreatic volume and bicarbonate secretion were observed. In these experiments, somatostatin did not influence secretin-stimulated bicarbonate concentration or rate of secretion but did influence the basal secretion of amylase. The CCK-stimulated enzyme and volume secretion were inhibited by somatostatin.

8703 SULPHATE METABOLISM IN THE EXOCRINE PANCREAS. II. THE PRODUCTION OF SULPHATED MACROMOLECULES BY THE MOUSE EXOCRINE PANCREAS. (Eng.) Berg, N. B. (State Univ. New York, Upstate Medical Center, Syracuse, NY 13210). *J. Cell Sci.* 31:199-211; 1978.

The types of sulfated macromolecules, in particular, proteins, proteoglycans, and lipids, produced by the exocrine mouse pancreas were investigated in pancreatic tissue taken from male Swiss mice. The behavior of sulfated macromolecules (labeled with ^{35}S-sulfate) and secretory proteins (labeled with ^{3}H-leucine) was compared. In the presence of 5×10^{-4} M cycloheximide, a strong inhibitor of protein synthesis, tissue uptake of radioleucine was reduced about 95% of control values during the first 30 min of incubation. A similar reduction in radiosulfate utilization was not evident until 4 hr of incubation, when an apparent intracellular pool of protein needed for the production of sulfated macromolecules was exhausted. Pilocarpine-induced secretion of macromolecules was studied by radioassay. In the presence of pilocarpine nitrate (5×10^{-7} M), both radioleucine- and radiosulfate-labeled secretory material attained a level of 40% net secretion at 90 min postpulse. Material labeled with inorganic sulfate consistently appeared in the chase medium 10-15 min prior to the secretory proteins, reflecting a shorter intracellular transport route taken by the sulfated material. In the next 90 min, the secretory proteins accumulated to nearly 70% net secretion while sulfated material accumulation reached only 50% net secretion. These differences in secretion show that leucine and inorganic sulfates are used for the production of different macromolecular species and indicate that the sulfated material is not

secretory protein. The radioactivity of lipid and proteoglycan fractions prepared from pancreatic tissue 4 hr after i.p. injection of radiosulfate demonstrated that both sulfated proteoglycans and sulfolipids were produced in the pancreas. The experiments demonstrate that pancreatic acinar cells can utilize inorganic sulfate for the sulfination of proteoglycans and lipids but not of proteins.

8704 ACUTE HEMORRHAGIC PANCREATIC NECROSIS IN MICE: BILE ACIDS IN GALLBLADDER BILE, SERUM AND PANCREAS. (Eng.) Yousef, I. M.; Rao, K. N.; Lombardi, B.; Fisher, M. M. (Dept. Medicine, Univ. Toronto, Toronto, Ontario M5S 1A8, Canada). *Acta Hepatogastroenterol.* 25(1):55-61; 1978.

Because bile acids are implicated in the pathogenesis of acute hemorrhagic pancreatic necrosis with fat necrosis, the bile acid composition of the pancreas, serum, and gallbladder bile of mice was studied. Mice were fed (a) a choline-deficient ethionine-supplemented (CDE) diet (0.5% DL-ethionine), (b) a choline-supplemented diet (CS), (c) a choline-deficient (CD) diet, or (d) a normal control diet for 1, 2, or 3 days. In addition, bile acids were analyzed in the pancreas, gallbladder, and serum of animals with pancreatic necrosis. Moderate degrees of compression atrophy of the mucosa and wall of the gallbladder were present in 2 of 12 mice fed the CS diet, in 5 of 14 mice fed the CD diet, and in 8 of 16 fed the CDE diet; severe degrees of atrophy were present in 5 of 12 fed the CS diet, in 9 of 14 fed the CD diet, and in 8 of 16 fed the CDE diet. The concentration of total bile acids in gallbladder bile of mice fed the CDE diet was significantly lower than that in mice fed the other diets ($p<0.05$). The amount of total bile acids in the gallbladders of mice fed the experimental diets was two- to fourfold greater than that in the controls. The bile acid composition of the bile was similar in mice on the normal and choline-supplemented diets. Choline deficiency reduced the relative amount of cholic acid, while ethionine supplementation increased the relative amount of the dihydroxy unsaturated cholanic acid. Plasma bile acid composition was affected by the different diets in a manner similar to that in the bile. However, the total bile acid concentration in the plasma of mice fed the CDE diet was significantly lower than that in the plasma of mice fed the other diets ($p<0.05$). In the pancreas, cholic acid accounted for only about 25% of the bile acids extracted, and no muricholic acids were found. Total bile acid concentration and bile acid composition were not affected by any of the diets. However, the amount of total bile acids per pancreas was significantly greater in mice fed the CDE diet ($p<0.05$), due to a larger pancreas weight in these animals, than that in the mice fed the other diets. Bile acids apparently do not initiate or participate in the extensive cellular damage preceding and accompanying the onset of acute hemorrhagic pancreatic necrosis with fat necrosis in mice fed a CDE diet.

8705 SPECIES SPECIFICITY AND OTHER ASPECTS OF CHYMOTRYPSIN INHIBITION BY PLASMA. (Eng.) Wolgemuth, R. L.; Hanson, K. M. (Ohio State Univ. Coll. Medicine, 333 W. Tenth Ave., Columbus, OH 43210). *Digestion* 16(1/2):96-107; 1977.

8706 HUMAN PANCREATIC CATIONIC TRYPSIN AS A POSSIBLE MEDIATOR OF PANCREATIC SHOCK AS INFERRED FROM A STUDY OF ITS INTERACTION WITH SERUM AND α_2-MACROGLOBULIN. (Eng.) Aubry, M.; Travis, J.; Bieth, J. (U.E.R. des Sciences Pharmaceutiques, Universite Louis Pasteur, 67083 Strasbourg, France). *Biochem. Med.* 18(3):360-370; 1977.

8707 IMPAIRED ENTERIC DEGRADATION OF PANCREATIC ENDOPEPTIDASES IN ANTIBIOTIC-TREATED RATS. (Eng.) Genell, S.; Gustafsson, B. E. (Malmo General Hosp., S-214 01 Malmo, Sweden). *Scand. J. Gastroenterol.* 12(7):801-809; 1977.

8708 EXTRAPANCREATIC GLUCAGONS. (Eng.) Holst, J. J. (Bispebjerg Hosp., DK-2400 Copenhagen NV, Denmark). *Digestion* 17(2):168-190; 1978.

See also, 8633, 8774, 8795, 8798, 9053, 9072, 9073, 9225.

SECRETION AND METABOLISM

Hepatobiliary Tract

8709 MICROSOMAL DRUG METABOLISM IN ACUTE AND CHRONIC GALACTOSAMINE INCUCED HEPATIC INJURIES. (Eng.) Shigeta, Y. (Sch. Medicine, Keio Univ., Tokyo, 160 Japan). *Gastroenterol. Jpn.* 12(6):435-445; 1977.

Female Wistar rats were given D-galactosamine (GalN) acutely (400 mg/kg body weight, i.p.) or chronically (250 mg/kg body weight daily for 7 months, i.p.) to determine its effects on the biochemical components and drug-metabolizing activities of the microsomal

membrane. Acute or chronic GalN administration produced significant ($p<0.01$) increases in liver weight compared with controls. Hepatic triglyceride concentration was significantly ($p<0.001$) increased by acute but unaffected by chronic GalN administration. The microsomal protein content was unchanged in the acute study but was significantly ($p<0.01$) decreased in the chronic study. Both studies demonstrated significant ($p<0.05$) increases in microsomal phospholipid content, expressed per mg of microsomal protein. Acute GalN administration produced a significant decrease in the cytochrome P450 content whether expressed per mg of microsomal protein ($p<0.001$) or per unit of body weight ($p<0.01$). Chronic GalN administration produced a significant increase in cytochrome P450 content when expressed per mg of microsomal protein ($p<0.01$) or per unit of body weight ($p<0.001$). Cytochrome b5 content was significantly increased in the acute study ($p<0.001$–0.05). NADPH cytochrome C reductase activity was unchanged in either study. The *N*-demethylating activity for aminopyrine was significantly decreased in the acute study when expressed per mg of microsomal protein ($p<0.02$) or per g of liver ($p<0.001$). In the chronic study, it was significantly increased when expressed per unit of body weight ($p<0.05$). Both studies demonstrated marked reductions in microsomal glucose-6-phosphatase activity. Electron microscopy of chronically damaged livers showed relatively well-preserved intracellular organelles of hepatocytes and an apparent proliferation of mitochondria. The smooth endoplasmic reticulum showed focally accentuated hypertrophy with liposomes in the dilated tubules. The data indicate that, in acute liver injury, hepatic microsomal drug-metabolizing activity is impaired. In chronic injury, the decreased drug-metabolizing capacity per unit of liver may be compensated by the enlarged hepatic mass, resulting in normal or even enhanced drug-metabolizing activity when expressed per unit of body weight.

8710 SIGNIFICANCE OF ENDOTOXAEMIA IN EXPERIMENTAL "GALACTOSAMINE-HEPATITIS" IN THE RAT. (Eng.) Grun, M.; Liehr, H.; Rasenack, U. (Medizinische Universitatsklinik, Josef-Schneider-Str. 2, 8700 Wurzburg, W. Germany). *Acta Hepatogastroenterol.* 23(2):64-81; 1976.

Normal, colectomized, and endotoxin-resistant rats were studied after the induction of hepatitis by an i.p. injection of GalN (1.0 g/kg) to determine whether endotoxins contribute significantly to the pathogenesis of galactosamine (GalN)-induced hepatitis. Endotoxemia was detected by the limulus gelation test (LGT) within 9 hr after GalN administration in the normal rats. Weak endotoxemia was present in only 25% of the colectomized animals and in none of the rats resistant to exogenous and endogenous endotoxins. Phagocytic capacity decreased significantly ($p<0.0001$) by 72 hr following GalN administration in the normal rats but not in colectomized or endotoxin-resistant rats. The development of other clinical symptoms of GalN-induced hepatitis, including pyrogen reaction, disseminated intravascular coagulation, arterial hypotension, and hypoglycemia, correlated significantly with the development of endotoxemia. Hyperbilirubinemia was present after GalN treatment only in the animals that did not undergo colectomy or procedures that made them endotoxin-resistant. Transaminase elevation was lower or normal in colectomized or endotoxin-resistant animals than in animals that did not undergo pretreatment. The results indicate that endotoxins contribute significantly to the pathogenesis of GalN-induced hepatitis and to its clinical symptoms.

8711 PROTECTIVE ACTIVITY OF WHITE PHOSPHORUS ON HEPATIC TOXICITY IN THE RAT. (Fre.) Quilichini, R.; Bildet, J.; Saurel, J.; Gendre, P. (Laboratoire de pharmacologie, Universite de Bordeaux II, 24 rue Paul Broca, 33000 Bordeaux, France). *Rev. Fr. Gastroenterol.* (138):25-36; 1978.

The ability of small doses of white phosphorus (WP) to prevent or reverse CCl_4-induced (1 g/kg, i.p.) liver toxicity was studied in male Wistar rats. Simultaneously, one-third of the rats received i.p. injections of a 10^{-14} dilution of 7 CH WP, one-third received injections of 10^{-30} 15 CH WP, and one-third received physiological serum (controls). The levels of SGOT, SGPT, and serum lactate dehydrogenase (LDH) were measured for 4 days after the injection. Histological studies on the livers of sacrificed animals were performed by electron microscopy 2, 12, and 48 hr after injection and by light microscopy 3, 8, and 15 days after injection. At 48 hr, the SGOT, SGPT, and LDH levels were consistently lower in rats given the 10^{-14} dilution of WP, compared with the animals treated with the 10^{-30} dilution of WP and the controls; the postinjection curves of the levels of SGOT, SGPT, and LDH were almost identical in the latter two groups over the 4-day period. Electron microscopy revealed fewer changes in the hepatocytes, such as degeneration and vacuolization, and light microscopy showed fewer areas of necrosis in rats treated with WP compared to controls; the lower dose of WP appeared histologically more efficacious. In a second study, rats were injected with two doses/day i.p. of either 10^{-14} or 10^{-30} dilutions of WP or with physiological serum for 15 days. These injections were discontinued 2 days prior to CCl_4 injection (1 g/kg, i.p.). The levels of SGOT, SGPT, and serum LDH, in particular, were lower in rats treated with either WP dilution compared with controls; the 10^{-14} dilution of WP was more efficacious than the 10^{-30} one in lowering liver enzyme levels after CT intoxication. Again, histological studies showed less evidence of necrosis in WP-treated rats. It is concluded that very weak dilutions of WP have a preventative and curative action with respect to the hepatic toxicity of CCl_4 in the rat.

8712 BILIARY SPASMOLYTIC ACTION OF 3-(2,4,5-TRIETHOXYBENZOYL) PROPIONIC ACID (AA-149) IN DOGS. (Eng.) Satoh, H.; Maki, Y.; Kanno, M. (Medicinal Res. Lab., Central Res. Div., Takeda Chemical Industries, Ltd., Osaka 532, Japan). *Eur. J. Pharmacol.* 48(3):309-317; 1978.

A study of 3-(2,4,5-triethoxybenzoyl)propionic acid (AA-149) was undertaken to clarify its mode of action on the motor function of the biliary tract in anesthetized dogs relative to autonomic drugs, gastrointestinal hormone, morphine, and taurocholate. The resistance of the choledochoduodenal junction (CDJ), gallbladder pressure (GP), bile flow, biliary ductal pressure, and blood pressure were recorded simultaneously. Doses included i.v. injections of carbachol (0.5 µg/kg), morphine (5 µg/kg), atropine (0.1-1 mg/kg), isoproterenol (1 µg/kg), propranolol (2-10 mg/kg), cholecystokinin (CCK, 0.1-1 mg/kg), and taurocholate (10 mg/kg). AA-149 was administered intrajejunally or i.v. at doses of 2, 5, or 10 mg/kg. At all dose levels and by both means of administration, AA-149 produced a dose-dependent reduction in the resistance of the CDJ and in GP along with a dose-dependent increase in bile flow. Like CCK, AA-149 decreased biliary ductal pressure while increasing the bile flow in dogs with ligation of the cystic duct of the gallbladder. Taurocholate produced increases in both biliary ductal pressure and bile flow. The spasmic response of the CDJ to morphine was not consistently depressed by atropine, but it was depressed strongly by AA-149. The effects of AA-149 were not influenced by pretreatment with atropine, phentolamine, or propranolol. The findings suggest that AA-149 relaxes the biliary tract (relaxation of the sphincter of Oddi) and depresses morphine-induced spasm by a mechanism of action different from that of atropine. The relaxing effect of AA-149 on biliary smooth muscle is not mediated by cholinergic or adrenergic mechanisms. It is likely that AA-149 acts directly on biliary smooth muscle rather than acting indirectly through release of CCK. The authors conclude that AA-149 is a novel compound with choleretic activity and with a strong spasmolytic activity on the biliary tract and that it acts by a mechanism different from those of anticholinergic and sympathomimetic agents.

8713 THE INFLUENCE OF β-SITOSTEROL ON BILIARY CHOLESTEROL SATURATION AND BILE ACID KINETICS IN MAN. (Eng.) Begemann, F.; Bandomer, G.; Herget, H. J. (I. Medizinische Klinik der Universitat Hamburg, Martinistr. 52, D-2000 Hamburg 20, W. Germany). *Scand. J. Gastroenterol.* 13(1):57-63; 1978.

To further investigate β-sitosterol as a possible agent for the treatment of cholelithiasis, its effect on cholesterol saturation of human bile and on bile acid kinetics was studied in 10 patients without hepatic or intestinal disease, 6 of whom had mild to moderate hyperlipemia. The administration of β-sitosterol (12 g/day, p.o.) for 6-8 weeks resulted in a mean decrease of 11% in serum cholesterol level ($p<0.02$) and of 12% in biliary cholesterol saturation ($p<0.05$); the latter effect was due to a change in the relative amounts of biliary lipids (lower cholesterol levels and higher bile acid values), whereas phospholipids varied insignificantly. Individual long-term observations revealed a sustained reduction or a further decrease in the lithogenic index in three patients, while in a fourth, who presented originally with undersaturated bile, an initial slight decrease of the lithogenic index was no longer detectable after 20 months. Significant changes in bile acid kinetics were not detected. The results lend support for further investigations of sitosterol as a possible adjunct therapy in cholelithiasis.

8714 EFFECT OF SPIRONOLACTONE ON BILE FORMATION AND LIVER MICROSOMAL ENZYMIC ACTIVITIES IN THE RAT. (Fre.) Miguet, J. P.; Mavier, P.; Arondel, J.; Preaux, A. M.; Dhumeaux, D. (Hopital Henri-Mondor, F 94010 Creteil, France). *Gastroenterol. Clin. Biol.* 1(12):1007-1013; 1977.

To elucidate the mechanism of the diuretic spironolactone on drug metabolism, the effect on bile formation, liver microsomal oxidation, and conjugation of drugs was studied in rats. Male Sprague-Dawley rats were given 15 mg/100 g spironolactone p.o. for 4 days and then fasted for 1 day without water restriction. After catheterization of the bile duct, bile was collected every 10 min for 30 min. The 14 treated rats had a 40% increase in bile flow, compared with the 18 control rats, entirely due to an enhancement of the bile salt independent fraction. The flow of bile salts (expressed in nmol/min/100 g) did not differ significantly in treated and control rats. Ten control and 10 treated rats were killed after the bile flow studies and their livers were weighed and homogenized for study of the enzymes in the microsomal fraction. Livers of diuretic-treated rats were significantly ($p<0.02$) heavier than those of control rats. Ethylmorphine demethylase, aminopyrine demethylase, and aniline hydroxylase activities were 18, 25, and 37% greater in treated rats than in the control rats ($p<0.05$, $p<0.05$, and $p<0.02$, resp.). UDP-glucuronyltransferase activity toward phenolphthalein, *p*-nitrophenol, and bilirubin was increased 38, 45, and 170% by the diuretic treatment ($p<0.02$, $p<0.02$, and $p<0.002$, resp.). Cytochome P-450 activity was not significantly altered in spironolactone-treated rats compared to controls. It is concluded that the effects of spironolactone on bile flow and hepatic microsomes could account for attenuation of the pharmacological effect of various drugs.

8715 EFFECT OF Na^+ ON BILE ACID UPTAKE BY ISOLATED RAT HEPATOCYTES: EVIDENCE FOR A HETEROGENEOUS SYSTEM. (Eng.) Anwer, M. S.; Hegner, D. (Fachbereich Tiermedizin der Universitat Munchen, Munchen, W. Germany). *Hoppe Seylers Z. Physiol. Chem.* 359(2):181-192; 1978.

The effect of Na^+ on cholate (C) and taurocholate (TC) uptake by isolated rat hepatocytes was investigated, to further characterize the Na^+ dependency of hepatic bile acid uptake and to test the hypothesis that hepatic bile acid uptake occurs through a multicarrier transport system. Uptake rates of both C and TC were decreased significantly when Na^+ was replaced by K^+, Li^+, or choline, suggesting that part of the bile acid uptake is Na^+-dependent. The Na^+-independent uptake rate of C (50-60%) was about

three times higher than that of TC. Since the specific cation used to replace Na^+ did not affect the Na^+-independent uptake rate significantly as the cation was varied, Na^+ was replaced by choline in all other experiments. Above 200 μM C or TC, the initial bile acid uptake rates increased linearly both in the presence and absence of Na^+. The slope of this "nonsaturable" component showed no Na^+ dependency, making it possible to calculate the Na^+-dependent uptake rate by subtracting the Na^+-independent uptake rate from the uptake rate in the presence of Na^+. Hyperbolic curves were obtained for Na^+-dependent uptake of both bile acids. Subtraction of the nonsaturable component from the Na^+-independent uptake rate also resulted in hyperbolic curves for both C and TC. These results indicate that both bile acids are transported into hepatocytes by (a) a nonsaturable uptake process, (b) a Na^+-independent saturable uptake process, and (c) a Na^+-dependent saturable uptake process. The inhibition of bile acid uptake by KCN (2 mM) and Oligomycin (20 μg/ml) was as follows: saturable Na^+-independent C uptake was inhibited by 60 ± 4% and 57 ± 5%, resp., saturable Na^+-dependent C uptake by 55 ± 4% and 30 ± 2%, resp., Na^+-independent TC uptake by 65 ± 6% and 44 ± 3%, resp., and Na^+-dependent TC uptake by 57 ± 4% and 29 ± 3%, resp. Uptake by the two saturable components was also inhibited 33-100% by other bile acids or analogues. These results suggest the operation of carrier-mediated, energy-requiring processes. The relationship between Na^+ concentration and the Na^+-dependent uptake rate was sigmoidal. The maximal uptake rate, rather than the K_m, was affected by Na^+, suggesting that more than one Na^+ act as cosubstrates in the transport of one bile acid molecule and that Na^+ exerts a stimulatory effect on the translocation of the bile acid-carrier complex across the membranes. Na^+-dependent uptake was inhibited by ouabain (0.1 or 1.0 mM), suggesting that this uptake process is linked to the activity of membrane-bound (Na^+-K^+) ATPase. Inhibition and competition studies suggested that C and TC share a common Na^+-independent transport system, while there are at least two Na^+-dependent transport systems for TC, one of which is shared by C.

8716 BILE ACID CONJUGATION IN ORGAN CULTURE OF HUMAN FETAL LIVER. (Eng.) Haber, L. R.; Vaupshas, V.; Vitullo, B. B.; Seemayer, T. A.; de Belle, R. C. (Montreal Children's Hosp., 2300 Tupper St. C-1220, Montreal, Quebec H3H 1P3, Canada). *Gastroenterology* 74(6):1214-1223; 1978.

An organ culture system for prolonged maintenance of human fetal liver was developed and used to investigate the formation of bile acid conjugates. Multiple liver specimens were obtained from human abortuses and stillbirths ranging from 10 to 29 weeks of gestation. Within 3 hr of hysterotomy, small fragments of liver were established in organ culture. The morphological integrity of the explants was demonstrated by light and electron microscopy. Functional viability was determined by adding radiolabeled primary bile acid to the medium and assaying the taurine and glycine conjugates formed. Primary bile acid was taken up by the tissues, conjugated with taurine and glycine, and secreted into the medium at a constant rate during 10 days *in vitro* and in constant increments during a selected 24-hr period. Human fetal liver survived intact for periods up to 10 days *in vitro*. Taurine conjugates of the primary bile acids were seen to predominate throughout gestation, and the mean glycine to taurine ratio was 0.32. Conjugates of cholic acid were synthesized in preference to those of chenodeoxycholic acid ($p<0.05$). The glycine to taurine ratio was significantly greater ($p<0.001$) when chenodeoxycholate was used as substrate compared with cholate in liver of the same gestational age. There was a tendency for the glycine to taurine ratio to increase with advancing gestational age ($p<0.01$). Supplementation of the medium with 3.75×10^{-4} M taurine resulted in enhanced taurocholate formation ($p<0.001$) with a concomitant decrease in glycocholate synthesis ($p<0.001$), suggesting competitive inhibition. This finding suggests that there is one acyl transferase system for both taurine and glycine. In medium supplemented with 2×10^{-6} M hydrocortisone, there was a reversal of the glycine-taurine ratio, which increased from a mean of 0.49 in the control group to a mean of 1.74 ($p<0.001$) in the experimental group.

8717 BILE ACID ESTER SULFATION IN THE HAMSTER AND MAN. (Eng.) Galeazzi, R.; Kok, E.; Javitt, N. B. *In: Bile Acid Metabolism in Health and Disease. Proceedings of the IV Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-12, 1976.* (Baltimore: University Park Press): 71-73; 1977.

To determine the sites of bile acid synthesis, the proportion of ester sulfates in the plasma of hamsters that underwent bile duct ligation was compared with that in hamsters that underwent either bilateral renal pedicle or bilateral ureteral ligation. In addition, serum bile acids were analyzed in hepatitis B surface antigen (HBsAg)-positive patients. The proportions of serum cholate and chenodeoxycholate in the hamsters that underwent bile duct ligation were similar to those in the animals that underwent either renal pedicle or ureter ligation, indicating that considerable ester sulfation occurs in animals having no circulation to the kidneys. Total serum bile acids in the patients with HBsAg ranged from 18 to 37 μg/ml, and the proportions of chenodeoxycholate and cholate were approximately the same. In two patients with advanced renal disease, the proportion of sulfates in the serum was greater than that found in similar individuals with normal renal function. The results in hamsters indicate that there is considerable extrarenal sulfation of bile acids, probably in the liver. In man, the association of increased ester sulfates of bile acids in plasma in advanced renal disease may also imply an extrarenal source for their synthesis.

8718 CONJUGATION AND SULFATION OF LITHOCHOLIC ACID IN FETAL LIVER. (Eng.) Watkins, J. B.; Brown, E. R. *In: Bile Acid Metabolism in*

Health and Disease, Proceedings of the V Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-12, 1976. (Baltimore: University Park Press): 65-70; 1977.

To determine whether the fetal liver is capable of sulfating lithocholic acid, thus rendering it less toxic and less readily reabsorbed by the intestine, in a manner similar to that found in the adult liver, bile acids were extracted from meconium from 15 newborn infants. In addition, experiments were carried out in a sheep model. Sulfated and nonsulfated bile acid conjugates were identified by gas-liquid chromatography and gas-liquid chromatography-mass spectroscopy. Of the bile acids isolated, 40-60% were found in the sulfate fractions. The majority of the secondary bile acids isolated were as sulfated derivatives, with nearly 60% of the sulfates being lithocholic acid principally as glycolithocholic acid sulfate, with the remainder being either glycodeoxycholic or glycoursodeoxycholic acid. Cholic and chenodeoxycholic acids were isolated predominantly in the nonsulfate fraction and principally as their taurine conjugates. Similar results were obtained in a fetal sheep model. Tracer doses of 24-^{14}C-24-lithocholate were administered i.v. to five chronic fetal sheep preparations, which were sacrificed 6-53 hr later. Maternal tissues contained 23 ± 4.6% of the recovered dose of ^{14}C-lithocholic acid injected into the fetus. The distribution of the label recovered in the mother was 78.5 ± 9.4% as taurolithocholate, 9.0 ± 3.6% as glycolithocholate, and 10.1 ± 5.2% as taurolithocholic acid sulfate. In the fetus, 91.4% of the lithocholate was conjugated with taurine and secreted into bile or intestine with a mean of 9.0% having been sulfated, the percentage of sulfation increasing with time. These experiments demonstrate that considerable quantities of sulfated lithocholic acid and other maternally derived secondary bile acids are found in the intestinal contents of the human infant at birth. The fetus may excrete and possibly detoxify maternally derived lithocholic acid either by excretion across the placenta or by the hepatic uptake, conjugation, sulfation, and then secretion into the fetal intestine. The high concentration of sulfated conjugates in meconium indicate that sulfation may represent a truly excretory process for the fetus and that these bile acids are not reabsorbed from the fetal intestine. Glycine-conjugated lithocholate may be sulfated more readily than the taurine-conjugated or the free bile acid.

8719 METABOLISM OF BILE ACIDS. V. METABOLISM OF URSODEOXYCHOLIC ACID IN RHESUS MONKEY. (Eng.) Ota, M.; Matsumoto, N.; Kuramoto, T.; Hoshita, T. (Hiroshima Univ. Sch. Medicine, Hiroshima, Japan). *Hiroshima J. Med. Sci.* 26(4):253-262; 1977.

The metabolism of p.o.-administered, radiolabeled ursodeoxycholic acid was studied in rhesus monkeys. After 24-[^{14}C]ursodeoxycholic acid administration (30 mg/kg), the maximum plasma level was observed at 30 min in two animals and at 8 hr in one monkey; the levels decreased biphasically in all animals thereafter. Most radioactivity was excreted in the feces, with transient elimination in the urine on the first day after administration. The biological half-life of ursodeoxycholic acid was approximately 2 days. Ursodeoxycholic acid in the liver was metabolized to tauroursodeoxycholate and glycoursodeoxycholate, which were excreted with the bile into the intestine. The taurine/glycine conjugate ratio in the bile varied with time after administration. The glycine conjugate predominated in the first 24 hr, and its proportion decreased thereafter; the proportion of the taurine conjugate excreted in bile increased in a complementary fashion. The conjugated bile acids were further hydrolyzed to free ursodeoxycholic acid, most of which was converted into lithocholic acid by intestinal microorganisms. Lithocholic acid was the major radioactive bile acid in the feces, but made up only a minor portion of biliary bile acids. Because of this, it seems likely that the lithocholic acid formed in the colon was poorly absorbed.

8720 SOME OBSERVATIONS ON THE MORPHOLOGICAL EVIDENCE FOR MECHANISM OF THE BILE SECRETION. (Eng.) Shin, Y. C. (Coll. Medicine, Korea Univ. 4, 2nd St., Myungyun-Dong, Chongno-Ku, Seoul, South Korea). *Acta Anat.* 100(4):499-511; 1978.

Bile secretion was studied ultrastructurally in the goldfish liver. The hepatocytes were seen to be closely packed between the sinusoids. Duct cells were embedded between the hepatocytes. The hepatocytes contained bile canaliculi. The lumina of these bile canaliculi were nearly obstructed with numerous microvilli. The mitochondria were similar in shape and distribution to those of mammalian hepatocytes. Lysosomes were usually present, and many of them consisted of homogenous material. These dense bodies showed acid phosphatase activity. The rough endoplasmic reticulum (RER) appeared as parallel arrays of flattened cisternae. The RER contained fibrillar material with occasional granules of low or moderate electron density. The smooth endoplasmic reticulum (SER) was especially prevalent in the vicinity of the Golgi apparatus. These smooth-surfaced tubules sometimes appeared to be in continuity with the RER. Smooth tubules in the vicinity of the Golgi apparatus were associated with linearly sacculated tubules. Isolated vacuoles containing cloudy material similar to that within the sacculated tubules and within the bile canaliculi were observed between the Golgi apparatus and the intracellular bile canaliculus and seemed to be derived from the linearly sacculated tubules associated with the Golgi apparatus. Some vacuoles were fused with the luminal cytolemmas of the bile canaliculus. It is concluded that these vacuoles participate in the transport of bile constituents. The duct cells showed no ultrastructural evidence to suggest that they are involved in bile secretion.

8721 EFFECT OF BILE SALTS ON THE BILIARY EXCRETION OF GLYCODIHYDROFUSIDATE IN THE

RAT. (Eng.) Montet, J. C.; Gerolami, A. (Unite de Recherches de Pathologie Digestive, INSERM, 46, Bd. de la Gaye, 13009 Marseille, France). *Can. J. Physiol. Pharmacol.* 56(2):163-167; 1978.

The biliary elimination of glycodihydrofusidate (GDHF), a structural analogue of bile salts, was studied in bile fistula rats to determine if GDHF and bile salts compete for biliary excretion or if bile salts increased GDHF transport as they do for BSP. GDHF was excreted in bile with a maximal excretory rate (T_m=0.80 μmol/min/kg) much lower than that of bile salts. When an i.v. bolus injection of 3 μmol of GDHF was followed after 2 min by a continuous dehydrocholate perfusion (10 μmol/min/kg), biliary excretion of GDHF was increased 24% in comparison with that in control rats. When the biliary T_m had been attained by continuous perfusion of GDHF at a rate of 1.35 μmol/min/kg, taurocholate or dehydrocholate infusion increased this T_m to a similar degree. The effects of dehydrocholate and taurocholate on GDHF biliary secretion suggest a stimulatory effect of bile salts on canalicular excretion of the drug. The results are similar to those previously obtained concerning the effects of bile salt infusions on the T_m of BSP and suggest that hepatic transport of GDHF and bile salts occurs by routes that are distinct for canalicular transport in spite of the striking structural similarities between GDHF and bile salts.

8722 BRAN AND BILE: TIME-COURSE OF CHANGES IN NORMAL YOUNG MEN GIVEN A STANDARD DOSE. (Eng.) Wicks, A. C. B.; Yeates, J.; Heaton, K. W. (Bristol Royal Infirmary, Bristol, England). *Scand. J. Gastroenterol.* 13(3):289-292; 1978.

The biliary response to a standard dose of bran was evaluated in 12 healthy men, and the time-course of previously observed changes in bile acid levels and biliary cholesterol saturation produced by dietary bran was investigated. All subjects added 15 g of bran to their usual diet and underwent aspiration of bile rich duodenal contents at 0, 1, 2, 4, and 6 weeks. In the 6-week sample, but not earlier, there was a significant decrease in the amount of deoxycholate (from 22.7 ± 2.8% to 16.5 ± 1.8%, $p<0.02$) and an increase in that of chenodeoxycholate (from 30.1 ± 1.6% to 34.9 ± 2.2%, $p<0.02$). Bile was unsaturated with cholesterol at the beginning of the experiment and remained so throughout. The delay in the action of bran on deoxycholate may occur because it reduces the formation of deoxycholate in the colon and does not merely impede its absorption.

8723 BILIRUBIN METABOLISM: STATE OF THE ART. (Eng.) Schmid, R. (Univ. California, San Francisco, HSW 1120, San Francisco, CA 94143). *Gastroenterology* 74(6):1307-1312; 1978.

Recent chemical and biochemical observations concerning bilirubin metabolism are reviewed. Aspects of bilirubin structure, with particular emphasis on the various pigment isomers and the importance of intramolecular hydrogen bonding are considered, and the hepatic conjugating mechanism and bilirubin conjugates are discussed. The conjugating mechanism of the liver cell seems to serve the sole purpose of altering the involuted hydrogen-bonded conformation of bilirubin IX by esterifying its two propionic acid groups with carbohydrate moieties. Only the bilirubin isomers that can form intramolecular hydrogen bonds, as in the natural bilirubin IX_α, require conjugation for excretion. Other isomers, including the artificial isomers IX_β, γ, and δ, and probably the photochemically-produced E isomers of bilirubin IX_α, can be excreted directly in bile.

8724 EFFECTS OF SATURATED AND UNSATURATED FATS GIVEN WITH AND WITHOUT DIETARY CHOLESTEROL ON HEPATIC CHOLESTEROL SYNTHESIS AND HEPATIC LIPID METABOLISM. (Eng.) Bochenek, W.; Rodgers, J. B. (Albany Medical Coll., Albany, NY 12208). *Biochim. Biophys. Acta* 528(1):1-16; 1978.

The effects of both dietary cholesterol and of fatty acid composition of the diet on hepatic cholesterol synthesis were studied in rats. Cholesterol synthesis was evaluated by measuring 3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMCoA) activity and by determining the rate of ^{3}H-labeled sterol production from ^{3}H-mevalonate. The results were correlated with sterol balance data and hepatic lipid content. The mean total hepatic lipid levels in rats fed a diet containing either saturated or unsaturated fat and no cholesterol were similar to those in controls fed a normal rat-chow diet. The total hepatic lipid levels in rats fed a high-cholesterol diet (1% of diet) were significantly higher ($p<0.001$) than the control level, and the highest levels (700 ± 153 mg total lipids) were found in the rats fed the high-cholesterol and unsaturated fat diet as opposed to the levels (453 ± 74 mg) in those fed the high-cholesterol and saturated-fat diet. The mean total hepatic cholesterol level in rats fed the low-cholesterol and saturated-fat diet was significantly lower than that in controls (23 ± 4.0 versus 28 ± 4.2 mg, $p<0.05$); the levels in rats fed the high-cholesterol and saturated-fat diet (50 ± 19 mg, $p<0.005$) and in rats fed the high-cholesterol and unsaturated-fat diet (124 ± 42 mg, $p<0.001$) were significantly higher than the control level. Fecal sterol outputs were similar in rats consuming either saturated or unsaturated fats. HMCoA activity was significantly higher in rats fed the low-cholesterol diet than in those fed the high-cholesterol diet both when the dietary fat was saturated (673 ± 358 versus 175 ± 91 pmol/mg protein/min, $p<0.001$) and when the dietary fat was unsaturated (446 ± 329 and 62 ± 14 pmol/mg/min, $p<0.002$). The levels of HMCoA activity were significantly lower in all animals fed the saturated-fat diet compared with controls ($p<0.05$), in those fed the high-cholesterol and unsaturated-fat diet compared with those fed the high-cholesterol and saturated-fat diet ($p<0.002$), and in those fed the unsaturated-fat diet compared with the controls ($p<0.005$ for high-cholesterol diet and $p<0.1$ for

low-cholesterol diet). ^{3}H-labeled sterol production from ^{3}H-mevalonate was significantly higher in rats fed the low-cholesterol diet than those fed the high-cholesterol diet both when the dietary fat was saturated ($p<0.05$) and when it was unsaturated ($p<0.001$). Sterol synthetic activity was significantly lower in animals fed the saturated fat diets ($p<0.005$ for low cholesterol, $p<0.001$ for high cholesterol) and in those fed the unsaturated fat diets ($p<0.001$) than in the controls. Cholesterol consumption decreased the percentages of essential polyunsaturated fatty acids. It is concluded that dietary cholesterol alters cholesterol and fatty acid metabolism in the liver and that this effect is enhanced by dietary unsaturated fats.

8725 EFFECTS OF CHOLESTEROL FEEDING ON SYNTHESIS AND METABOLISM OF CHOLESTEROL AND BILE ACIDS IN GERMFREE RATS. (Eng.) Gustafsson, B. E.; Angelin, B.; Einarsson, K.; Gustafsson, J. A. (Karolinska Institutet, Stockholm, Sweden). *J. Lipid Res.* 18(6):717-721; 1977.

The influence of cholesterol feeding on cholesterol synthesis and fecal excretion of bile acids was studied in germfree rats. Four germfree rats were fed a basal diet containing 0.004% cholesterol, and four germfree rats received the same diet supplemented with 0.4% cholesterol for 2 weeks. Cholesterol synthesis was studied by assaying the 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase activity in the liver microsomal fraction. Cholesterol feeding decreased the mean HMG CoA reductase activity from 28.5 ± 6.6 to 9.1 ± 0.7 pmol/mg protein/min. In another experiment, four germfree rats received the basal diet and four germfree rats, the cholesterol-enriched diet. After 6 weeks, feces were collected in two 4-day pools for analysis of bile acids. The main fecal bile acids were cholic acid and β-muricholic acid, comprising more than 95% of total bile acids. Cholic acid was increased from 3.9 ± 0.2 to 9.9 ± 1.2 mg/kg/day and β-muricholic acid from 6.6 ± 0.5 to 21.8 ± 3.1 mg/kg/day. The percentage of cholic acid decreased from 37.1 ± 1.1 to 31.2 ± 1.0%. Germfree rats, like conventional rats, have the ability to compensate for an increased input of dietary cholesterol by inhibition of cholesterol synthesis and stimulation of bile acid synthesis. The synthesis of chenodeoxycholic acid (inferred from the fecal excretion of β-muricholic acid) is stimulated to a greater extent than that of cholic acid.

8726 IMPAIRMENT OF HEPATIC CHOLESTEROL SYNTHESIS FROM SQUALENE AND THE FUNCTION OF HEPATIC STEROL CARRIER PROTEIN SYSTEM BY AGEING. (Eng.) Takeuchi, N.; Koga, M.; Yamamura, Y.; Tanaka, F.; Yamaguchi, Y.; Uchida, K. (Osaka Univ. Hosp., Fukushima-ku, Osaka, Japan 553). *Exp. Gerontol.* 13(1/2):1-7; 1978.

Cholesterol synthesis from squalene was measured in adult and in developing male Sprague-Dawley rats, and the effects of cytosol fractions from different age-groups on cholesterol synthesis of the hepatic microsomes from respective groups were compared *in vitro*. Incorporations of squalene and mevalonate into cholesterol in hepatic microsomes and cytosol obtained from growing (4-week-old) or young (2-month-old) rats were higher than those in microsomes and cytosol from adult rats. When the microsomes from growing or young rats were combined with the cytosol from adult rats instead of with young cytosol, the incorporation rates of squalene and mevalonate into cholesterol were decreased. When adult microsomes were combined with young cytosol, cholesterol synthesis from mevalonate was increased, but that from squalene was not. Cytosol fractions from cholesterol-fed rats did not inhibit cholesterol synthesis from squalene in the microsomes from nontreated rats, and the normal cytosol did not influence cholesterol synthesis in the microsomes from cholesterol-fed rats, suggesting that the elevation of serum cholesterol in aged animals did not affect the activity of the cytosol fraction. It is concluded that the function of the hepatic sterol carrier protein is probably impaired by ageing process.

8727 THE EFFECT OF AGEING ON THE BILIARY EXCRETION OF OUABAIN IN THE RAT. (Eng.) Kitani, K.; Kanai, S.; Miura, R.; Morita, Y.; Kasahara, M. (Tokyo Metropolitan Inst. Gerontology, Itabashi-ku, Tokyo, Japan). *Exp. Gerontol.* 13(1/2): 9-17; 1978.

Studies were carried out to gain information regarding age-related changes in hepatic elimination and biliary excretion of ouabain in the rat. The plasma concentration and biliary excretion of ouabain were determined in 3-, 6-, 16-, 18-, and 24-month-old male Wistar rats that each received an i.v. injection of ^{3}H-ouabain (0.4 mg/100 g). The plasma ouabain concentration increased with age, an effect most clearly seen in plasma samples taken at 2 and 5 min after injection. In 3-month-old rats, the level was 9.3 ± 1.1 µg/ml at 2 min and 5.1 ± 1.9 µg/ml at 5 min; in 24-month-old rats, it was 19.9 ± 2.4 µg/ml and 9.1 ± 1.0 µg/ml, resp. The difference was greater between 3- and 6-month-old rats than between 6- and 24-month-old rats. As time elapsed after ouabain injection, the age difference in plasma concentration decreased. The biliary excretion rate of ouabain was highest in the first 10-min bile sample in 3-month-old rats, and the excretion rate decreased thereafter. In contrast, excretion in 6-month-old or older rats was lower in the first sample, and the peak excretion was observed in the second 10-min sample. In the first 10-min sample, the excretion rate of 24-month-old rats was less than one-third of that in 3-month-old rats. Spironolactone pretreatment (7.5 mg/100 g/day) for 4 days in 16-month-old rats accelerated biliary ouabain excretion by an average of approximately 30%, in comparison with that in controls. The cumulative excretion rate in these rats approached the level seen in 6-month-old rats. The results indicate that there are age-related differences in ouabain elimination. These differences may be due to changes in bile flow rate.

8728 IN VITRO AMINO ACID TRANSPORT INTO BRAIN REGIONS OF RATS WITH A PORTACAVAL ANASTOMOSIS. (Ita.) Rigotti, P.; Pizzolato, G.; Zanchin, G.; Bonino, M.; Vassanelli, P.; Battistin, L. (Istituto di Patologia Speciale Chirurgica, Padova, Italy). *Acta Chir. Ital.* 33(3):486-492; 1977.

A preliminary study of the uptake of amino acids by slices of forebrain, cerebellum, mesodiencephalon, and pons medulla of rats is presented. Male Sprague-Dawley rats were subjected to portacaval shunts; 1 month later, when the rats had a hunchbacked appearance and lowered reactions to stimuli, they were decapitated, and their brains were divided into four parts. Slices made from each of the four parts were placed into oxygenated medium and incubated 1 hr with four ^{14}C-labeled amino acids, then homogenized and analyzed for radioactivity. A decreased uptake of all four amino acids was revealed when compared with uptake by brain slices from unoperated controls. Glutamic acid uptake in comparison with controls was -4% in forebrain, -9% in cerebellum, -15% in mesodiencephalon, and -7% in pons medulla. The results for gamma-aminobutyric acid were -16% in forebrain ($p<0.10$), -2% in cerebellum, -10% in mesodiencephalon, and -9% in pons medulla. Glycine uptake was -22% in forebrain ($p<0.01$), -5% in cerebellum, -8% in mesodiencephalon, and -2% in pons medulla. Valine uptake was -16% in forebrain ($p<0.02$), -15% in cerebellum, -21% in mesodiencephalon ($p<0.02$), and -25% in pons medulla ($p<0.001$). These observations suggest that cerebral transport is affected in these experimental animals by portacaval anastomosis. The possible role of transport mechanisms impaired by portasystemic encephalopathy deserves further study.

8729 SOME OBSERVATIONS ON THE LIVER AND LUNG-MICROSOMAL-HYDROXYLATING ENZYME-ACTIVITY IN PORTO-CAVAL SHUNTED RATS. (Eng.) Vassanelli, P.; Richter, C. (Istituto di Patologia Chirurgica dell'Universita di Padova, Policlinico, via Giustiniani, 35100 Padova, Italy). *Acta Chir. Ital.* 33(4):608-612; 1977.

Since the lungs and liver have the same embryological origin from primitive gut and since the presence of some microsomal drug metabolizing enzyme activities in the lungs has been established, liver and lung drug metabolism was examined in rats with portacaval shunt. P-nitroanisol demethylase activity was significantly increased in the shunted rats (0.54 nmol/min/mg protein) compared with that in controls (0.29 nmol/min/mg), whereas no significant differences in cytochrome P-450 content and NADPH-cytochrome c reductase activity were noted after shunt. Benzo(α)pyrene hydroxylase activity in microsomes isolated from the lungs of rats with portacaval shunt was also increased. It appears that portacaval shunt may alter drug metabolism in rats, and that drug administration in patients with a portacaval shunt should be approached with caution.

8730 THE RABBIT AS AN EXPERIMENTAL ANIMAL FOR THE STUDY OF PORTASYSTEMIC ENCEPHALOPATHY (PRELIMINARY DATA). (Ita.) Vassanelli, P.; Battaglia, G.; Merigliano, S.; Zannini, P. (Istituto di Patologia Chirurgica dell'Universita di Padova, Padova, Italy). *Acta Chir. Ital.* 33(3):478-485; 1977.

The use of rabbits to study portasystemic encephalopathy is advocated and discussed. Fifty rabbits were given end-to-side portacaval shunts, performed below the gastroduodenal vein to maintain the blood flow to the liver. They were maintained on diets differing in protein content (19, 7.6, or 14.9% of the dry material). Body weight and electroencephalograms were studied. The average weight of the 10 rabbits subjected to the shunt was 2.1 kg at the time of the operations and 1.6 kg 10 days after the operations. On day 10 after the operations, 10 operated rabbits and 10 that had been sham-operated were decapitated, and their brains were analyzed for gamma-aminobutyric acid (GABA) levels. Rabbits given a standard diet (19% protein) died within 10 days, and those given either diet of hay survived as long as 2 months. In all the rabbits surviving the operation, central nervous system disturbances (incoordination and difficulty in walking) were observed. The animals showed evidence of exaggerated amounts of fear, a tendency to bang their heads against their cages, and altered electroencephalograms. In all the operated rabbits, the GABA levels were raised an average of 273 γ% mg of brain compared with an average of about 98 γ% mg of brain in the controls. It is concluded that the rabbit makes an interesting experimental model for portasystemic encephalopathy because the size of the animal yields large tissue and blood samples, neuropathy is manifest in a short period of time after the operation (7-10 days), and increased levels of cerebral GABA are detectable following the operation.

8731 SYNERGISTIC ACTION OF HEPATOCYTE MEMBRANE DEFECT AND ACTIVATED COMPLEMENT SYSTEM IN LIVER CELL DEATH: AN EXPERIMENTAL APPROACH TO FULMINANT HEPATIC FAILURE. (Eng.) Liehr, H.; Grun, M.; Seelig, H. P.; seelig, R.; Rasenack, U. (Medizinische Universitatsklinik, Josef-Schneider Str. 2, D-8700 Wurzburg, W. Germany). *Acta Hepato-gastroenterol. (Stuttg.)* 25(2):105-110; 1978.

The hypothesis that massive liver cell necrosis is caused by activation of the complement system as a result of a hepatocyte membrane defect was tested in Chbb Thom strain rats. The animals received 50, 100, 200, or 1,000 mg/kg galactosamine i.p. with or without the simultaneous i.v. injection of 1.5 mg/kg endotoxin. Doses of 50 mg/kg or 100 mg/kg galactosamine produced no liver cell necrosis, whether or not endotoxin was administered simultaneously. The 200 mg/kg dose led to moderate liver cell necrosis, and when administered together with endotoxin, the animals developed

fulminant hepatic necrosis. Animals that received 1 g/kg galactosamine together with endotoxin died approximately 12 hr later, whereas animals receiving 1 g/kg galactosamine alone were alive at 24 hr after administration. In the animals that received endotoxin and the high dose of galactosamine, immunohistological examination revealed that the third complement component was already fixed on the hepatocyte plasma membranes at 3 hr after administration and was accumulated within areas of necrotic parenchymal cells at 12 hr. It appears that liver cell death is complement-mediated if the hepatocyte membranes are altered.

8732 SURGICALLY INDUCED HEPATIC FAILURE IN ANIMALS. (Eng.) Ouchi, K.; Nose, Y.; Hermann, R. E. (Cleveland Clinic, Cleveland, OH). *Cleve. Clin. Q.* 44(4):171-180; 1977.

Three types of surgically-induced hepatic failure (anhepatic, ischemic, and jaundiced) were induced in mongrel dogs (4 dogs/group) to examine the usefulness of each of these animal models of hepatic failure for understanding the underlying mechanisms of this entity and developing human therapeutic modalities. The animals in the anhepatic group survived 26, 17, 17, and 11 hr after the total hepatectomy, and the postoperative course was similar in all four. The clinical observations (vomiting of gastric contents and coffee-groundlike material; spastic motions; clonic convulsions; shallow and rapid breathing succeeded by deep and slow breathing; decline in systolic blood pressure, pulse rate, and respiratory rate; and oliguria succeeded by anuria) and the biochemical observations (severe acidosis and hypercapnea; respiratory alkalosis but decrease in arterial pH; decrease in all blood cell components; decrease in serum Na^+ and Cl^- levels; increase in serum K^+ level; gradual decreases in total protein, albumin, and fibrinogen levels; increases in all liver function tests; decline of cholesterol and blood urea nitrogen levels; increase in creatinine, uric acid, ammonia, and free fatty acid levels; slight decrease in the levels of branched-chain amino acids; moderate increase in phenylalanine and tyrosine; and marked increase in glutamic acid, glycine, and histidine levels) were extremely similar to those in humans with hepatic insufficiency. The animals in the ischemic group underwent occlusion of the common hepatic artery either at the same time, 48 hr after (2 animals), or 6 days after portacaval anastomosis; one of the two animals in the second group did not develop coma, so that ligation of the proper hepatic artery was also carried out (6 days after portacaval anastomosis). The onset time of coma and the survival time after the last procedure were directly related to the time interval between portacaval anastomosis and hepatic artery ligation; onset of coma occurred at 7, 12, 14, and 35 hr, resp., and survival time was 16, 18, 18, and 38 hr, resp., for these animals. The biochemical changes were very similar to those in the anhepatic model except for higher levels of the nonbranched-chain amino acids. Following operations to induce obstructive jaundice, the remaining four animals survived 5, 5, 7, and 17 weeks after surgery, resp. As is frequently observed in patients with obstructive jaundice, the causes of death (and therefore the clinical and biochemical observations) varied; the dogs died of severe liver abscess with renal failure, pneumonia, gastric hemorrhage from multiple erosions, and hepatic failure from biliary cirrhosis, resp. It is concluded that these animal models closely resemble hepatic insufficiency in humans.

8733 BILIVERDIN INITIATES THE LIVER REGENERATION IN THE RAT--A HYPOTHESIS. (Eng.) Okazaki, K.; Nishimura, H.; Arizono, H.; Nishimura, N.; Suzuki, Y. (Aichi Medical Univ. Sch. Medicine, Nagakute, Aichi, Japan). *Biochem. Biophys. Res. Commun.* 81(2):512-520; 1978.

The hypothesis that biliverdin initiates liver regeneration was investigated in intact, sham-operated, and 90% hepatectomized female Wistar rats. Biliverdin was absent in the plasma of intact and sham-operated animals, while the levels in hepatectomized animals killed at 5, 10, 20, 30, 45, 60, 120, and 180 min after ligating the blood vessels and bile ducts serving the right lateral and caudate lobes were 3.2, 2, 1.3, 1.3, 2, 1.7, 0.7, and 0.7 μg/ml, resp. In another series of experiments, the values were 4.2, 0.7, 0.7, 1.3, 1, 1.7, 0.7, and 0.3 μg/ml, resp. The mitotic indices determined in 100,000 hepatic parenchymal cells from intact rats at 24, 80, and 32 hr after a single i.p. injection of biliverdin (10 mg/100 g) were 446 ± 118, 862 ± 68, and 224 ± 8, resp. Mitotic indices determined 30 hr after the i.p. injection of 0.1 mg/100 g biliverdin, bilirubin, or hemoglobin were 130 ± 47, 129 ± 2, and 26 ± 2, resp. After a 1 mg/100 g injection, the levels were 92 ± 5, 106 ± 1, and 87 ± 5, resp., while the levels after a 10 mg/100 g injection were 862 ± 68, 20 ± 8, and 681 ± 134, resp. The results support the hypothesis that biliverdin initiates liver regeneration in rats.

8734 CELLULAR ANALYSIS OF LIVER CELL NECROSIS INDUCED BY ACETAMINOPHEN [Abstract]. (Eng.) Shinozuka, H.; Lincoln, S.; Iammarino, R. M.; Vogt, M. T. (Sch. Medicine and Health Related Professions, Univ. Pittsburgh, PA 15261). *Fed. Proc.* 37(3):402; 1978.

8735 MECHANISM OF ETHANOL-DRUG INTERACTION IN RAT HEPATOCYTES [Abstract]. (Eng.) Chengelis, C. P.; Schulman, M. P. (Univ. Illinois Coll. Medicine, Chicago, IL 60612). *Fed. Proc.* 37(3):568; 1978.

8736 ACETALDEHYDE-INDUCED LIPID PEROXIDATION IN ISOLATED HEPATOCYTES [Abstract]. (Eng.) Stege, T. E.; Daniels, S. E.; Sturtz, J. (Ohio Wesleyan Univ., Delaware, OH 43015). *Fed. Proc.* 37(3):303; 1978.

8737 ACUTE EFFECTS OF ETHANOL ON MEMBRANES OF ENDOPLASMIC RETICULUM OF RAT LIVER [Abstract]. (Eng.) Murty, C. N.; Verney, E.; Sidransky, H. (George Washington Univ. Medical Center, Washington, DC 20037). *Fed. Proc.* 37(3):318; 1978.

8738 BIOCHEMICAL ALTERATIONS IN ORGANS OF MICE INFECTED WITH Hymenolepis microstoma, THE MOUSE BILE DUCT TAPEWORM. (Eng.) Pappas, P. W. (Coll. Biological Sciences, Ohio State Univ., Columbus, OH 43210). *J. Parasitol.* 64(2):265-272; 1978.

8739 EFFECT OF DIAMFENETIDE ON EXPERIMENTAL INFECTIONS OF Fasciola hepatica IN LAMBS: ANTHELMINTIC AND CLINICAL INVESTIGATIONS. (Eng.) Rew, R. S.; Colglazier, M. L.; Enzie, F. D. (Animal Parasitology Inst., U.S. Dept. Agriculture, Beltsville, MD 20705). *J. Parasitol.* 64(2):290-294; 1978.

8740 A METHOD FOR GALLBLADDER BILE COLLECTION WITHOUT THE NEED OF A THOMAS CANNULA. (Eng.) Soto, J. M.; Wallen, A. E.; Dreiling, D. A. (Mount Sinai Sch. Medicine, Fifth Ave. and 100 St., New York, NY 10029). *Mt. Sinai J. Med.* 45(1):116-117; 1978.

8741 REFLUX-FREE BILIARY TRACT RECONSTRUCTION USING A CANINE ILEOCOLONIC SEGMENT. (Eng.) Berlatzky, Y.; Lebensart, P.; Freund, H. (Hadassah Univ. Hosp., Jerusalem, Israel). *Surg. Gynecol. Obstet.* 146(5):768-772; 1978.

8742 THE EFFECT OF ALIPHATIC ALCOHOLS ON CERTAIN VITAMINS OF THE B-COMPLEX GROUP IN THE LIVER OF THE RAT. (Eng.) Shehata, M.; Saad, S. (Dept. Pharmacology, Univ. Cairo, Cairo, Egypt). *Pol. J. Pharmacol. Pharm.* 30(1):35-39; 1978.

8743 ACTIVE ENHANCEMENT OF CANINE LIVER ALLOGRAFTS WITH POLYSPECIFIC ANTIGEN. (Eng.) Lie, T. S.; Rao, G. S.; Ueda, T.; Kim, B. R.; Rommelsheim, K. (Medical Faculty, Univ. Bonn, Bonn, W. Germany). *Surg. Gynecol. Obstet.* 146(5):783-788; 1978.

8744 SIZE AND BLOOD FLOW OF THE LIVER ESTIMATED BY $^{99}Tc^{m}$ SCANNING. (Eng.) Pirttiaho, H. I.; Pitkanen, U. (Dept. Internal Medicine, Univ. Oulu, SF-90220 Oulu, Finland). *Acta Radiol. Ther. Phys. Biol.* 16(6):497-506; 1977.

8745 AN ELECTRON MICROSCOPIC STUDY OF EXPERIMENTAL CHRONIC VIRAL HEPATITIS IN NUDE MICE. (Eng.) Kakudo, K.; Sakurai, M.; Takahashi, S.; Kitamura, H. (Osaka Univ. Medical Sch., Osaka, Japan). *Med. J. Osaka Univ.* 28(1):57-66; 1977.

8746 EFFECT OF INTERRUPTION OF HEPATIC ARTERY ON LIVER REGENERATION. (Eng.) Ohtoshi, E.; Mizumoto, R.; Honjo, I. (Kyoto Univ. Medical Sch., Kyoto, Japan). *Arch. Jpn. Chir.* 46(6):681-688; 1977.

8747 STRAIN DIFFERENCE IN MOUSE CHOLELITHIASIS AND THE EFFECT OF TAURINE ON THE GALLSTONE FORMATION IN C_{57}BL/C MICE. (Eng.) Fujihira, E.; Kaneta, S.; Ohshima, T. (Hokkaido Inst. Pharmaceutical Sciences, Katsuraokacho-62, Otaru, Japan). *Biochem. Med.* 19(2):211-217; 1978.

8748 LIVER CELL HETEROGENEITY: THE DISTRIBUTION OF FRUCTOSE-BISPHOSPHATASE IN FED AND FASTED RATS AND IN MAN. (Eng.) Schmidt, U.; Schmid, H.; Guber, W. G. (Medizinischen Poliklinik der Universitat Basel, Hebelstr. 1, CH-5046 Basel, Switzerland). *Hoppe Seylers Z. Physiol. Chem.* 359 (2):193-198; 1978.

8749 PERSISTENT INFECTION WITH MOUSE HEPATITIS VIRUS OF LOW VIRULENCE IN NUDE MICE. (Eng.) Tamura, T.; Taguchi, F.; Ueda, K.; Fujiwara, K. (Inst. Medical Science, Univ. Tokyo, 4-6-1, Shiroganedai, Minato-ku, Tokyo 108, Japan). *Microbiol. Immunol.* 21(12):683-691; 1977.

8750 EFFECT OF GLUCAGON AND INSULIN ON THE GROWTH OF NEONATAL RAT HEPATOCYTES IN PRIMARY TISSUE CULTURE. (Eng.) Armato, U.; Draghi, E.; Andreis, P. G. (Istituto di Anatomia Umana Normale, Via A. Gabelli 65, I-35100, Padova, Italy). *Endocrinology* 102(4):1155-1166; 1978.

8751 ALCOHOL AND HEPATIC DRUG METABOLISM. (Fre.) Pirotte, J. (Institut de Medecine, Universite de Liege, Liege, Belgium). *Acta Gastroenterol. Belg.* 40(7/8):278-283; 1977.

8752 COMPARATIVE STUDIES ON ORTHOTOPIC LIVER ALLOTRANSPLANTATION IN PIGS AFTER INTOXICATION WITH EXTRACTS FROM Amanita phalloides AND AFTER A LONG-TERM PRESERVATION OF TRANSPLANTS. (Eng.) Koristek, V.; Cerny, J.; Gregor, Z.; Rokl, J. (II. Chirurgische Universitatsklinik der J. E. Purkyne-Universitat, 656 02 Brno, Czechoslovakia). *Z. Exp. Chir.* 10(6):384-390; 1977.

8753 CHOLELITHIASIS IN A MALE RHESUS MONKEY (Macaca mulatta) FED A CHOLESTEROL-CONTAINING DIET. (Eng.) Geistfeld, B. L.; Bond, M. G.; St. Clair, R. W. (Bowman Gray Sch. Medicine, Wake Forest Univ., Winston-Salem, NC 27103). *J. Med. Primatol.* 6(4):237-244; 1977.

8754 EFFECTS OF DIBUTYRYL ADENOSINE 3',5'-MONOPHOSPHATE ON HEPATIC METABOLISM OF

FREE FATTY ACIDS. (Eng.) Klausner, H. J.; Soler-Argilaga, C.; Heimberg, M. (Univ. Missouri Sch. Medicine, Columbia, MO 65201). *Metabolism* 27(1): 13-25; 1978.

8755 EVIDENCE FOR ADDITIONAL MEDIATOR IN PROSTAGLANDIN-INDUCED CHOLERESIS. (Eng.) Ruwart, M. J.; Kaminski, D.; Jellinek, M. (Dept. Surgery, St. Louis Univ., St. Louis, MO 63104). *Prostaglandins* 14(5):975-982; 1977.

8756 EFFECT OF DIETARY WHEAT GLUTEN ON LIPID METABOLISM IN GROWING RATS. (Eng.) Mokady, S.; Einav, P. (Dept. Food Engineering and Biotechnology, Technion-Israel Inst. Technology, Haifa, Israel). *Nutr. Metab.* 22(3):181-189; 1978.

8757 EFFECT OF PANTOTHENIC ACID DEFICIENCY ON RAT HEPATOCYTES. (Eng.) Mahboob, S.; Estes, L. W. (Sch. Medicine, Univ. Pittsburgh, Pittsburgh, PA 15261). *Nutr. Metab.* 22(3):177-180; 1978.

See also, 8624, 8704, 8776, 8777, 9079, 9084, 9095, 9106, 9165, 9172, 9176, 9177, 9181, 9217, 9226.

SECRETION AND METABOLISM

Intestine

8758 A MODIFIED PROCEDURE FOR THE RAPID PREPARATION OF EFFICIENTLY TRANSPORTING VESICLES FROM SMALL INTESTINAL BRUSH BORDER MEMBRANES: THEIR USE IN INVESTIGATING SOME PROPERTIES OF D-GLUCOSE AND CHOLINE TRANSPORT SYSTEMS. (Eng.) Kessler, M.; Acuto, O.; Storelli, C.; Murer, H.; Muller, M.; Semenza, G. (Laboratorium fur Biochemie, Eidgenossische Technische Hochschule, Universitatsstr. 16 and 2, CH-8092 Zurich, Switzerland). *Biochim. Biophys. Acta* 506(1):136-154; 1978.

A simplification of a previously described procedure for the preparation of brush border membranes from small intestine is described. The procedure was tested for Na^+-dependent D-glucose transport, and it was also used to study choline transport across the brush border membrane. The procedure is simple, rapid, does not necessarily require scraping, and can be started from fresh or frozen material. It can be scaled up easily, allowing a quick production of large amounts of brush border membrane vesicles. $CaCl_2$ (10 mM) is added to mucosal homogenates and allowed to stand in the cold for 15 min. The suspension is centrifuged at 3,000 x *g* for 15 min, and the supernatant is then centrifuged at 27,000 x *g* for 30 min. The pellet is resuspended in the 40 ml of buffer needed for the experiment, homogenized, and centrifuged at 27,000 x *g* for 30 min. The transport ability of the "calcium vesicles" obtained by these methods was compared with that of "EDTA vesicles" reported previously. The calcium vesicles constantly produced a longer lasting and higher overshoot peak of D-glucose (3-8 times as high as the equilibrium value with 1 mM D-glucose and 100 mM NaSCN as the initial outer concentration). Even NaSCN or NaCl gradients as low as 10 to 0 mM produced detectable overshoots. The calcium vesicles allowed the unequivocal demonstration of the transient accumulation of L-alanine, which was impossible with EDTA vesicles. After 2-3 days at 4 C, calcium vesicles generally showed only a minor decrease in their transport ability, demonstrating their stability. These results indicate that the calcium vesicles are excellently suited for transport studies. Choline transport was found to occur by a saturable component with a K_m of 83 ± 4 μM (at 20 C) and by a nonsaturable component. It was independent of the presence of Na^+, since it was not affected when K^+ was substituted for Na^+, and appeared to be nonelectrogenic. The charge movement associated with choline transport into these vesicles must be compensated by the exit of another cation or the entry of an anion, through the same transport agency as choline.

8759 DOES HEPARIN OCCUR IN MUCOSAL MAST CELLS OF THE RAT SMALL INTESTINE? (Eng.) Tas, J.; Berndsen, R. G. (Histological Lab., Univ. Amsterdam, 1e Const. Huygensstraat 20, Amsterdam, Netherlands). *J. Histochem. Cytochem.* 25(9):1058-1062; 1977.

The chemical nature of the glycosaminoglycan (GAG) moiety of rat mucosal mast cell granules was investigated using microspectrophotometric analyses combined with model experiments using polyacrylamide films containing different types of purified GAG or GAG and basic protein. For the microspectorphotometric recording of metachromatic curves from toluidine blue O (TBO)-stained mucosal mast cells, a staining pH of 1.0 was chosen to avoid interference by orthochromatic staining. The time course of the metachromatic peaks recorded from the TBO-stained mucosal mast cells after mounting in glycerol was plotted. An "equilibrium level," with a

mean peak wavelength of 553 ± 4.5 nm and 553 ± 4.1 nm for duodenal and jejunal mast cells, resp., was reached after about 6 hr. However, within 36 hr all mast cells in the sections had faded completely. When the same technique was applied to peritoneal and mesenteric mast cells, a final mean peak wavelength of 514 ± 3.5 nm and 516 ± 5.2 nm, resp., was obtained. The equilibrium level was reached after about 5 days in both cases, and metachromatically stained mast cells were present for about 8 days. Model experiments using polyacrylamide films that contained various types of GAGs showed that the metachromatic peaks recorded after staining at pH 1.0 instead of the more usual pH level of 5.0 differed only slightly. To imitate the chemical composition of rat mast cell granules more closely, model films containing GAG and chymotrypsinogen A were stained under the same conditions. After staining at pH 5.0, the metachromatic peaks shifted again to higher wavelengths on addition of this basic protein to the GAG in the model films. This shift was further increased by staining these films at pH 1.0. In all cases, the peak changes were most pronounced in glycerol. The peak of the metachromatic complex of TBO with heparin, the highest sulfated GAG used, was found to be less sensitive to the effects just mentioned. The results indicate that significant amounts of heparin do not occur in these cells and that lower sulfated GAG(s) are present in the granules.

8760 ROLE OF THE SMALL INTESTINE IN LIPID METABOLISM. SMALL INTESTINAL METABOLISM OF PLASMA FATTY ACIDS. (Ger.) Gangl, A. (II. Univ.-Klinik fur Gastroenterologie und Hepatologie, Garnisongasse 13, A-1090 Vienna, Austria). *Fortschr. Med.* 96(13):713-717; 1978.

The metabolism of plasma free fatty acids in the small intestine was studied by the i.v. injection of ^{14}C-labeled palmitic acid in rats and in 12 human volunteers undergoing suction biopsy of the small intestine. Two minutes after the injection of palmitic acid into rats, 1% of the radioactivity was found in the small intestinal mucosa, 42% in water soluble oxidation products (CO_2, keto acids, and other metabolites), and only 16% in triglycerides. Experiments with simultaneous i.v. injection of ^{14}C-palmitic acid and intraduodenal injection of ^{3}H-palmitic acid showed that the fatty acid introduced into the intestine was mainly esterified to triglyceride, while that injected i.v. was mainly oxidized and incorporated into phospholipids. An intraduodenal infusion of glucose (2 g/kg/hr), cholestyramine (5% suspension), taurocholate (10 and 30 mmol), or cholecystokinin (1.5 Ivy U/kg, s.c.) had no noteworthy effects on the intestinal metabolism of plasma free fatty acids, while intraduodenally administered alcohol (0.33 g/kg/hr) inhibited the intestinal oxidation of fatty acid by 60% and doubled both the palmitic acid incorporation into triglycerides by the intestinal mucosa and the serum triglyceride concentration. Intraduodenally administered oleic acid and monooleate (82 mg in a molar ratio of 2:1, emulsified in 10 mM taurocholate) doubled the intestinal uptake of plasma free fatty acids, quadrupled their incorporation into intestinal triglycerides, and caused a sixfold increase in the secretion of plasma free fatty acids into intestinal lymph triglycerides. Similar findings were also obtained in the volunteers. The findings indicate the great significance of the small intestine in lipid and lipoprotein metabolism.

8761 SPECIFIC LOCALISATION OF NEUROTENSIN TO THE N CELL IN HUMAN INTESTINE BY RADIOIMMUNOASSAY AND IMMUNOCYTOCHEMISTRY. (Eng.) Polak, J. M.; Sullivan, S. N.; Bloom, S. R.; Buchan, A. M. J.; Facer, P.; Brown, M. R.; *et al.* (Royal Postgraduate Medical Sch., London W12, England). *Nature* 270(5633):183-184; 1977.

To investigate the distribution of neurotensin in the human gastrointestinal tract, samples of normal stomach, intestines, gallbladder, and pancreas were obtained at surgery and examined using radioimmunoassay and immunocytochemistry. Large amounts of neurotensin-like reactivity were found in the ileal mucosa. The number of cells staining solely with antibodies to neurotensin diminished as the jejunum was reached, and they were extremely sparse in the duodenum. Neurotensin immunoreactivity was not detected in the stomach, pancreas, or colon by immunocytochemistry, but small amounts were detected by radioimmunoassay in the stomach (0.27 ± 0.03 pmol/g wet weight) and colon (0.50 ± 0.19 pmol/g). The amounts of immunoreactivity in the duodenal, jejunal, and ileal mucosae were found to be 0.24 ± 0.10, 2.82, and 16.2 pmol/g, resp., by this technique. No reactivity was found in the pancreas or gallbladder. The immunoreactive cells were located in the upper two-thirds of the villi and were often seen to communicate with the intestinal lumen by means of microvilli. When serial 1-μm sections were stained with antibodies reacting with enteroglucagon (EG) and vasoactive intestinal peptide (VIP), the positive cells were completely distinct from those stained by the neurotensin antibodies. The VIP- and EG-positive cells contained considerably smaller granules than did the neurotensin cells. These studies indicate that neurotensin cells occur in high concentrations in the ileal mucosa, with lesser quantities in the jejunum, duodenum, stomach, and colon. The morphological features of these cells suggest that they are sensitive to changes in the gut lumen, which may provide the stimulus for the release of the peptide directly into the circulation.

8762 BREAKDOWN OF MUCIN AND PLANT POLYSACCHARIDES IN THE HUMAN COLON. (Eng.) Vercellotti, J. R.; Salyers, A. A.; Bullard, W. S.; Wilkins, T. D. (Anaerobe Lab., Virginia Polytechnic Inst. and State Univ., Blacksburg, VA 24061). *Can. J. Biochem.* 55(11):1190-1196; 1977.

To obtain an estimate of the extent to which complex carbohydrates are degraded by bacteria in the human colon, aqueous extracts of colon contents from four human subjects were separated into high and low molecular weight fractions by chroma-

tography on a Sephadex G-100 column. The composition of these fractions was compared with the composition of similar fractions from ileal contents, i.e., from material entering the colon. There were two distinct peaks; a high molecular weight peak (approximately 100,000 daltons) and a low molecular weight peak (approximately 1,000 daltons) in all subjects. In all subjects, the concentration of high molecular weight carbohydrate in the ileum was more than twice the concentration of high molecular weight carbohydrate in the colon, indicating that breakdown of complex carbohydrate occurs in the colon. The high molecular weight carbohydrate fraction contained sugars characteristic of plant polysaccarides (arabinose, xylose, mannose, rhamnose) as well as sugars characteristic of mucin (fucose, hexosamines, sialic acids). Concentrations of most of these sugars were uniformly lower in the colon than in the ileum. Since high molecular weight protein concentrations were lower in the colon than in the ileum of two of the four subjects tested, some degradation of protein may also occur in the colon.

8763 TURNOVER OF RAT BRUSH BORDER PROTEINS AFTER MASSIVE INTESTINAL RESECTION. (Eng.) Fehlmann, M.; Starita-Geribaldi, M.; Isman, H.; Sudaka, P. (Laboratoire de Biochimie, Universite de Nice, Chemin de Vallombrose 06200, Nice, France). *Biochem. Biophys. Res. Commun.* 81(2):493-497; 1978.

To determine if the turnover of brush border membrane proteins is affected by massive intestinal resection, the relative degradation rates of brush border proteins were compared in male Wistar rats that had undergone 75% proximal small bowel resection with those in rats that underwent sham operation. After small bowel resection, there was a significant decrease in the $^{3}H:^{14}C$ ratios for high molecular weight (>70,000 daltons) brush border proteins when compared with proteins of similar size isolated from sham-operated animals ($p<0.05$). These results indicate that brush border membrane protein turnover is affected by 75% small intestinal resection. The authors conclude that this change may reflect immaturity of the villus epithelial cells in the remaining ileum after resection.

8764 IMMUNOCYTOCHEMICAL STUDY OF THE INTERACTION OF SOYBEAN TRYPSIN INHIBITOR WITH RAT INTESTINAL MUCOSA. (Eng.) Wilson, P. A.; Melmed, R. N.; Hampe, M. M. V.; Holt, S. J. (Ian Clunies Ross Animal Res. Lab., P.O. Box 239, Blacktown, New South Wales 2148, Australia). *Gut* 19(4):260-266; 1978.

To investigate further the cause of the pancreatic enlargement induced by orally ingested soybean trypsin inhibitor (STI), antibodies raised against STI and purified by affinity chromatography were used to localize dietary STI in the rat gut by fluorescent immunocytochemical methods. This technique permitted the clear intracellular demonstration of STI in the ileal mucosa of suckling rats; in adult rats no entry of STI into mucosal cells of the small intestine was demonstrated, being confined to the luminal surface of the mucosa. Although the passage of STI into and across the adult intestinal mucosa could not be excluded through the use of this technique, the results were consistent with a previously described intraluminal mode of action of STI, namely, that the pancreatic enlargement caused in sensitive species results from the inhibition of trypsin (which acts as the physiological inhibitor of the mucosal secretion of pancreotrophic hormones), thus resulting in the uninhibited secretion of these hormones.

8765 RELEASE OF VASOACTIVE INTESTINAL POLYPEPTIDE (VIP) BY INTRADUODENAL STIMULI. (Eng.) Schaffalitzky de Muckadell, O. B.; Fahrenkrug, J.; Holst, J. J.; Lauritsen, K. B. (Bispebjerg Hosp., Bispebjerg Bakke 23, DK-2400 Copenhagen NV, Denmark). *Scand. J. Gastroenterol.* 12(7):793-799; 1977.

To determine whether stimuli other than i.v. calcium or electrical stimulation of the vagal nerves will elicit the release of vasoactive intestinal polypeptide (VIP) into the circulation, the effects of intraduodenal infusion of amino acids, glucose, fat, HCl, ethanol, and saline on plasma VIP concentrations were investigated in seven normal human subjects and 12 anesthetized pigs. In addition, the plasma VIP concentration after the ingestion of a mixed meal was determined in six normal subjects, and the effect of HCl on plasma VIP concentration was determined in five postvagotomy patients. In normal subjects, the median peripheral blood plasma VIP concentration in the basal state was 4.3 pM (range 0-12.0 pM). No significant changes in the VIP concentration occurred after amino acid infusion (40 ml of a mixture of alpha and beta casein), glucose infusion (0.3 M, 40 ml), NaCl infusion (0.15 M or 1.5 M, 40 ml), or ingestion of a meal. In contrast, HCl (0.1 M, 40 ml), isotonic fat emulsion (20%, 40 ml), and ethanol (60 ml Vodka) infusion increased the plasma VIP concentration in all seven normal subjects; the mean peak values after HCl, fat, or ethanol were 9.8 (range 5.9-12.6), 7.5 (2.4-10.2), and 12.6 (7.8-16.8) pM, resp. Truncal vagotomy did not change the response produced by HCl, and the VIP level increased from 5.8 pM (range 0-11.6 pM) to 10.2 pM (7.5-13.0 pM) after HCl infusion in the vagotomized patients. The results from VIP determinations in the portal plasma of pigs following amino acid, glucose, fat, HCl, ethanol, or saline infusion confirmed the findings in the peripheral blood plasma of the normal human subjects and showed that the VIP levels in portal plasma were 1.6-2.9 times higher than the VIP levels in arterial plasma. The pH threshold to VIP release was 1.1-2.1, and the effect of HCl was not abolished by ganglionic blockade with hexamethonium (10 mg/kg). These studies show that intraduodenal HCl, fat, and ethanol stimulate VIP release. The lack of VIP response to a meal (which contained fat) may be explained by the fact that changes in VIP concentration are greater in the portal plasma than in the peripheral circulation: VIP

release might be detectable in the portal circulation only.

8766 EFFECT OF PENTAGASTRIN AND SECRETIN ON CELL RENEWAL AND THE THREE DIMENSIONAL STRUCTURE OF THE JEJUNAL MUCOSA OF THE FASTED RAT. (Fre.) Bosshard, A.; Pansu, D.; Lambert, R. (Hopital Edouard-Herriot, F 69374 Lyon Cedex 2, France). *Gastroenterol. Clin. Biol.* 2(2):165-176; 1978.

The effect of pentagastrin and secretin on the renewal of cells and the structure of the jejunal mucosa were studied in male Wistar rats fasted for 48 hr. In the second 24-hr period of fasting, the rats were injected i.p. with pentagastrin (175 µg/100 g) or secretin (24.5 clinical U/100 g) divided into seven doses given at 4-hr intervals. One group of hormone-treated animals was injected with tritiated thymidine (50 µCi/100 g) at the same time as the first injection of hormone and then killed either at the end of 48 or 72 hr. Animals from the second hormone-treated group were killed every 4 hr during the third 24-hr period, with tritiated thymidine injected 30 min before sacrifice. Control fasted rats had significantly ($p<0.05$) lower labeling and mitotic indices compared to nonfasted control rats; the wet weight of the intestines and the thickness of the intestinal wall were also decreased compared with nonfasted rats. In fasted rats injected with secretin, there was a decrease of the labeling index followed by a decrease of the mitotic index during the third 24-hr period compared with fasted control rats; also, the number of short villi in the intestinal mucosa increased. After the pentagastrin injections, labeling and mitotic indices increased until, at 72 hr, they were significantly ($p<0.05$) higher in treated rats than in fasted controls. Pentagastrin also induced thickening of the serosal villi and of the mucosal layer. The results confirm the trophic effect of gastrin on cell renewal and the three dimensional structure of the jejunum and the antitrophic effects of secretin on these parameters.

8767 EVALUATION OF THE EFFECTS OF CELLULOSE ON FECAL AND INTESTINAL CHARACTERISTICS OF MICE. (Eng.) Araujo, P. E. (Dept. Food Science & Human Nutrition, IFAS, Univ. Florida, Gainesville, FL 32611). *J. Food Sci.* 43(3):1040; 1978.

8768 FURTHER INVESTIGATIONS ON INDOMETHACIN AND INTESTINAL ULCERS IN THE RAT. (Eng.) Del Soldato, P.; Meli, A. (Menarini Pharmaceuticals, 50131 Florence, Italy). *Toxicology* 9(1/2):69-74; 1978.

8769 INHIBITION OF PROSTAGLANDIN BIOSYNTHESIS IN RAT SMALL INTESTINE BY SL-573. (Eng.) Yanagi, Y. (Pharmaceuticals Div., Sumitomo Chemical Co., Ltd., 4-2-1, Takatsukasa, Takarazuka, Hyogo, 665, Japan). *Biochem. Pharmacol.* 27(5):723-728; 1978.

8770 ENTEROTOXIC ACTIVITY OF Salmonella weltevreden CULTURE FILTRATES. (Eng.) Thapliyal, D. C.; Singh, I. P. (Dept. Microbiology and Public Health, G. B. Pant Univ. Agriculture & Technology, Patnagar, India). *Indian J. Exp. Biol.* 16(3):396-398; 1978.

8771 RELEASE OF GASTROINTESTINAL HORMONES INTO THE DUODENAL LUMEN OF CATS. (Eng.) Uvnas-Wallensten, K.; Efendic, S.; Luft, R. (Karolinska Inst., Stockholm, Sweden). *Horm. Metab. Res.* 10(2):173; 1978.

8772 EFFECT OF NITROGEN SOURCE ON NUTRITIONAL MANAGEMENT AFTER SMALL BOWEL RESECTION IN RATS. (Eng.) Kishi, T.; Iwasawa, Y.; Itoh, H.; Chibata, I. (Res. Lab. Applied Biochemistry, Tanabe Seiyaku Co., Ltd., Osaka 533, Japan). *J. Nutr. Sci. Vitaminol.* 24(2):167-176; 1978.

8773 MORPHOLOGICAL CHANGES IN THE SMALL INTESTINE OF THE CAT FOLLOWING TRANSECTION OF THE VAGAL NERVE. (Eng.) Feher, E.; Gasnyi, K.; Vajda, J. (Semmelweis Univ. Medical Sch., Tuzolto utca 58, 1450 Budapest, IX, Hungary). *Acta Anat. (Basel)* 101(3):218-224; 1978.

See also, 8620, 8626, 8630, 8640, 8641, 8681, 8722, 8738, 8775, 8776, 8778, 8949, 8994, 9052, 9175.

GENERAL

8774 RETROGRADE PANCREATIC PHLEBOGRAPHY WITH SIMULTANEOUS BALLOON OCCLUSION OF PORTAL AND ARTERIAL FLOW. AN EXPERIMENTAL STUDY. (Ger.) Gunther, R.; Fabsguth, A.; Georgi, M.; Hohn, P. (Institut fur klinische Strahlenkunde der Universitat, Langenbeckstrasse 1, 6500 Mainz, W. Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 128(2):151-155; 1978.

The possibility of visualizing the venous system of the entire pancreas by retrograde portal contract injection (with Angiografin) and simultaneous balloon occlusion of the portal vein, celiac trunk, and superior mesenteric artery was studied in 12 male dogs. Good filling of the major pancreatic veins was achieved in 7/12 dogs. The portal and arterial pressures altered considerably

under the experimental conditions. A rise in the levels of serum transaminases was observed only once, when there was thrombosis of the portal vein and branches of the hepatic veins. Apart from this case, there was no rise in the levels of transaminases or of α-amylase. After a total occlusion of 40 min (6-min periods of occlusion alternating with 2-5 min of flow), there were only minor histological changes in the liver. Short-term arterial balloon occlusion seems feasible in man as well, but a transumbilical approach should be chosen.

8775 BEHAVIOR OF SOME SERUM ENZYMES FOLLOWING LIGATION OF THE SUPERIOR MESENTERIC ARTERY IN THE RAT (PRELIMINARY REPORT). (Ger.) Ruprecht, H.; Gaida, P.; Keil, E.; Kunath, H.; Klett, U. (Chirurgische Universitatsklinik und Poliklinik, Friedrich-Schiller-Universitat, Bachstrasse 18, DDR-69 Jena, E. Germany). *Z. Exp. Chir.* 10(6):351-356; 1977.

To evaluate serum enzyme tests as diagnostic tools for acute intestinal ischemia, the effect of ligation of the superior mesenteric artery on some serum enzymes was studied in adult female Wistar rats 0.5-8 hr after ligation. The ligation of the superior mesenteric artery caused marked increases in the activities of the sérum enzymes (aspartate amino transferase, alanine amino transferase, lipase, alpha-amylase, isocitrate dehydrogenase, glutamate dehydrogenase) and in the blood sugar level. The aspartate amino transferase activity increased markedly with time between the 2nd and 8th hr, while the alanine amino transferase activity reached its maximum between 3 and 4 hr, the lipase activity between 1 and 3 hr, the alpha-amylase activity between 2 and 4 hr, and the glutamate dehydrogenase activity between 3 and 4 hr. The isocitrate dehydrogenase activity increased with time between the 2nd and 8th hr. The changes in the enzyme activities are probably due to the shock and to the reduced inflow of portal vein blood to the liver (and thus to additional hypoxic damage of this organ). The findings indicate that the serum enzyme tests may be valuable in the diagnosis of early stages of acute intestinal ischemia.

8776 EMBRYOLOGY OF THE SPHINCTER OF ODDI. (Eng.) Germain, M.; Martin, E.; Gremillet, C. *In: The Sphincter of Oddi. Proceedings of the Third Gastrointestinal Symposium, Nice, June 8-9, 1976.* (Basel: S. Karger): 1-5; 1977.

8777 CHYLOMICRON REMNANT PARTICLE FORMATION IN VIVO IN HEPATECTOMISED RAT. (Eng.) Vost, A.; Pocock, D. M. E.; Pleet, S. (McGill Univ. Medical Clinic, Montreal, Quebec, Canada). *Adv. Exp. Med. Biol.* 82:139-141; 1977.

8778 A STUDY OF THE MORPHOLOGY AND KINETICS OF EPITHELIAL MIGRATION IN RESPONSE TO GASTRO-INTESTINAL ULCERATION: A NEW APPROACH TO THE CANCER-ULCER QUESTION. (Eng.) Stemmermann, G. N.; Hayashi, T.; Taki, M. *In: Pathophysiology of Carcinogenesis in Digestive Organs. Proceedings of the 7th Symposium of The Princess Takamatsu Cancer Research Fund, Tokyo, 1976.* (Univ. of Tokyo Press): 37-47; 1977.

8779 SPONTANEOUS COLON CANCER IN RATS. (Eng.) Miyamoto, M.; Takizawa, S. *In: Pathophysiology of Carcinogenesis in Digestive Organs. Proceedings of the 7th International Symposium of The Princess Takamatsu Cancer Research Fund, Tokyo, 1976.* (Univ. of Tokyo Press): 297-304; 1977.

8780 PURIFICATION OF INFECTIOUS PANCREATIC NECROSIS (IPN) VIRUS AND COMPARISON OF POLYPEPTIDE COMPOSITION OF DIFFERENT ISOLATES. (Eng.) Chang, N.; MacDonald, R. D.; Yamamoto, T. (Dept. Microbiology, Univ. Alberta, Edmonton, Alberta T6G 2E9, Canada). *Can. J. Microbiol.* 24(1):19-27; 1978.

8781 STUDIES ON EXPERIMENTAL MIXED INFECTION OF Schistosoma haematobium AND Schistosoma mansoni IN THE ALBINO MOUSE. (Eng.) Halawani, A. A.; Farag, H. F.; Awadalla, H. N. (Medical Res. Inst., Alexandria, A.R.E. 165, El Horreya Av., El Hadara, Egypt). *Tropenmed. Parasitol.* 28(4): 478-480; 1977.

8782 CHARACTERIZATION OF THE IMMUNOCHEMICAL FORMS OF CALCITONIN RELEASED BY A MEDULLARY THYROID CARCINOMA IN TISSUE CULTURE. (Eng.) Goltzman, D.; Tischler, A. S. (Dept. Medicine, McGill Univ., Montreal, Quebec, Canada). *J. Clin. Invest.* 61(2):449-458; 1978.

See also, 8674, 8698, 9020, 9056.

8783 ARTERIOPORTOGRAPHY. (Eng.) Herlinger, H. (St. James' Univ. Hosp., Leeds, England). *Clin. Radiol.* 29(3):255-275; 1978.

The results of 82 arterioportographic (AP) examinations of 78 patients are described. Because splenoportography (S) can give inadequate or misleading information and because there are contraindications and serious potential complications associated with its use, AP has superseded it in the study of portal hypertension. There are no contraindications to AP, complications are totally avoidable, and the information obtainable comprises both the arterial and venous phases, encompasses the entire portal system, allows a limited estimate of flow dynamics, and contributes to planning for shunt surgery. The technique of AP involves the catheterization of the celiac and superior mesenteric arteries, of the inferior vena cava, and of the hepatic veins. In the cases reported, arterial phase findings included retroperitoneal malignancy (4), hepatoma complicating cirrhosis (5), pancreatitis, hepatic artery occlusion (underperfusion, 2), and a nontraumatic communication between the left hepatic artery and the left portal vein (1). Splenic artery catheterization and injection of an adequate amount of contrast medium, with good flow through the spleen, provided pictorial information virtually equal to that obtained by S. Venous phase information included morphological inadequacy (portal vein [PV], 2; splenic vein [SV], 4; superior mesenteric vein [SMV], 6), abnormality of flow (hepatofugal PV, 3; slow tidal SV, 8), occlusion (PV, 5; SV, 13; SMV, 5), and presence of thrombus (PV, 6; SMV, 11). Aids to interpretation of venous phase data are given with the warning that S should not be used to confirm the diagnosis of SV occlusion as hemorrhage from the spleen could occur. Wedged hepatic venography was discussed although it was not used formally in the current study to grade the severity of cirrhosis or to test for portal flow reversal. Criteria for the use or avoidance of mesocaval and distal splenorenal shunt procedures and for the use of percutaneous transhepatic portal catheterization and/or AP in the management of patients with portal hypertension are presented. The study demonstrates that AP is an angiographic method with comprehensive diagnostic potential, which is management oriented and useful for determining the suitability of patients for specific shunt surgery.

8784 CONCLUSIONS OF LP-X DETERMINATION IN MORE THAN 2,500 PATIENTS. (Eng.) Eder, G.; Weiss, W.; Neumayr, A.; Schuller, J.; Baumgartner, G. (Immuno Diagnostika, Vienna, Austria). *Protides Biol. Fluids Proc. Colloq.* 25:341-347; 1977.

The results of two different studies on the determination of serum lipoprotein X (LP-X) levels for the detection of extra- or intrahepatic cholestasis are presented. In one study, 800 healthy subjects were screened for LP-X, none of whom were found to have this factor in the serum. In the other study, 1,805 patients in a gastroenterology department were tested, 1,570 of whom had histologically confirmed hepatobiliary disease. The first LP-X test was positive in 28.9% of these patients. However, different results were recorded at follow-up 1 week later in 23.3% of 257 patients and at follow-up 2 weeks later in 51.5% of 68 patients. The LP-X test was positive in 92.8% of 181 cases of cholestasis. The test was positive in 77.6% of 165 patients with alcoholic liver diseases, in 63.7% of 91 patients with hepatitis B surface antigen (HBsAg)-positive acute hepatitis, in 50.0% of 66 patients with HBsAg-negative acute hepatitis, in 42.4% of 33 patients with chronic active hepatitis, and in 2.9% of 34 patients with chronic persistent hepatitis. LP-X was present in the sera of 44.4% of 54 patients with primary liver cancer, 48.8% of 170 patients with liver metastases, 5.1% of 99 patients with a tumor but no liver metastases, and in 23.3% of 194 patients with hematologic malignancies. LP-X occurred in 73.9% of 23 patients with osteomyelosclerosis but in less than 31% of other patients with hematologic diseases. The LP-X test was positive in 28.4% of 95 cases of cholelithiasis, 88.9% of 27 cases of cholelithiasis, and 35.0% of 40 patients with inflammatory bowel disease. LP-X testing is useful in the diagnosis of intra- and extrahepatic cholestasis.

8785 CHOLANGIOGRAPHIC APPEARANCE OF THE INTRAHEPATIC BILE DUCTS. (Eng.) Wojtowicz, J.; Karwowski, A. (Katedra Radiologii AM, ul. Szkolna 8/12, 61-833 Poznan, Poland). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 127(5):417-421; 1977.

The value of percutaneous transhepatic cholangiography (PTC) in the diagnosis of intrahepatic bile duct diseases was assessed by examining the cholangiographic appearance of the intrahepatic bile ducts in 134 cases. The patients underwent the procedure for gallstone disease (23 cases), evaluation of complications of gallbladder surgery (21), chronic pancreatitis (6), liver cirrhosis and chronic hepatitis (4), gallbladder or bile duct neoplasms (31), pancreas or papilla neoplasms (36), or liver neoplasms (13). Satisfactory filling of the peripheral bile ducts was obtained after drainage of bile and with the injection of a somewhat increased quantity of contrast medium into the biliary system. Generalized dilatation of the intrahepatic bile ducts was observed in all cases of mechanical obstruction. PTC was effective in detecting malignant neoplasms: the incidence of true positives was 36/40 and the incidence of true negatives was 89/94. The predictive value of a positive result was 36/41. Advanced liver cirrhosis with extrahepatic bile obstruction was characterized by PTC by dilatation with pronounced crowding and tortuosity of the bile ducts. Multiple abscesses associated with cholangitis were an incidental finding of PTC; these appeared as extravasations of contrast medium into the liver parenchyma communicating freely with the biliary system. The PTC appearance of surgical lesions of the choledochal duct, necrosis of a metastatic tumor, and of cystic dilatations of the peripheral branches of the biliary system are illustrated. These results show that PTC can be useful in the diagnosis of intrahepatic disease.

8786 PLASMA BILE ACIDS FRACTIONATION BY GAS-LIQUID CHROMATOGRAPHY: APPLICATION TO HEPATOCELLULAR DEFICIENCY INVESTIGATION. (Fre.) Bereziat, G.; Pepin, D.; Wolf, C.; Polonovski, J. (Laboratoire de Chimie Biologique, CHU Saint-Antoine, 27, rue Chaligny, 75571, Paris Cedex 12, France). *Pathol. Biol. (Paris)* 25(8):559-564; 1977.

The four major bile acids, cholic, chenodeoxycholic, deoxycholic, and lithocholic acids, were measured in plasma from patients with atresia of biliary tract (3), viral hepatitis (5), and cirrhosis (12), including alcoholic and posthepatic disease. The method used was that of Nair employing gas-liquid chromatography. Normal levels established in this series were compared with those reported by other authors. In the presence of cholestasis, the ratio of chenodeoxycholic acid to cholic acid was equal to or less than one; when hepatic deficiency developed, the ratio of chenodeoxycholic acid to chclic acid was higher than one. A very high ratio was considered indicative of progressive hepatic deficiency. Three patients with high levels of lithocholic acid had hepatic disease with a fatal outcome.

8787 COMPUTED TOMOGRAPHY OF THE GALLBLADDER. (Eng.) Havrilla, T. R.; Reich, N. E.; Haaga, J. R.; Seidelmann, F. E.; Cooperman, A. M.; Alfidi, R. J. (Cleveland Clinic Foundation, 9500 Euclid Ave., Cleveland, OH 44106). *Am. J. Roentgenol.* 130(6):1059-1067; 1978.

Clinical experience with computer tomography (CT) in 79 patients with suspected gallbladder disease is presented. CT accurately diagnosed stones within the gallbladder, either radiopaque or radiolucent, in 78% of 50 patients with cholelithiasis. There were three false-positive CT examinations. In one case, laminated calcifications in the renal parenchyma adjacent to the gallbladder were mistakenly identified as gallstones. One patient had chronic cholecystitis with a thickened gallbladder wall and adhesions, and the third had an inflammatory mass in the right liver lobe (no calculi). The 11 false-negative studies included 4 patients with a nonvisualized gallbladder; in 5 patients, calculi were missed completely. The CT scan correctly identified 6 of 7 patients with a functioning gallbladder without stones and 14 of 15 patients with nonfunctioning gallbladders without stones. Four patients without cholelithiasis but with either an abscess or gallbladder bed inflammation secondary to cholecystitis were all identified by the CT examination. One patient with a nonvisualizing oral cholecystogram and negative reading of a plain film of the abdomen had a large laminated calculus shown by CT, which appeared to be outside the confines of the gallbladder and situated in the portal area. At laparotomy, a gangrenous gallbladder was found with perforation of a large gallstone into the liver bed and porta hepatis. In two cases, the presence of hydrops secondary to cystic duct obstruction or to common bile duct stones were noted on the CT scan. The observations in these patients suggest that manifestations of gallbladder disease such as hydrops, opaque and nonopaque gallstones, chronic cholecystitis with thickened inflammatory walls, and secondary liver abscesses can be detected by CT. It is a useful technique for individuals in whom the gallbladder has failed to opacify on oral cholecystography, and can be a useful alternative to conventional radiographic or ultrasound examinations, giving an overall diagnostic accuracy greater than 80%.

8788 COMPUTED TOMOGRAPHY OF THE PANCREAS. (Eng.) Moss, A. A.; Kressel, H. Y. (Univ. California, Sch. Medicine, San Francisco, CA). *Am. J. Dig. Dis.* 22(11):1018-1027; 1977.

Experience with computed tomography (CT) in the diagnosis of pancreatic diseases is discussed, and numerous illustrations are presented. CT scanning demonstrates the normal pancreas in most cases. It is unusual to image the entire pancreas on a single CT scan. Except in extremely emaciated patients, a layer of peripancreatic fat separates the pancreas from the aorta and enables the superior mesenteric artery to be identified just posterior to the body of the pancreas. In detecting pancreatic abnormality, attention should be paid to alterations in morphologic configuration that produce diffuse or focal areas of pancreatic enlargement, and to nonhomogeneous pancreatic tissue that produces variations in pancreatic tissue density (CT number, attenuation coefficient). The most important sign of pancreatic carcinoma is focal enlargement of the head, body, or tail of the pancreas. When the tumor extends posteriorly, the peripancreatic fat plane may be obliterated by tumor extension. Acute pancreatitis causes either focal or more often diffuse enlargement of the pancreas. In some instances, the peripancreatic fat plane may be destroyed, and differentiation of localized pancreatic enlargement due to pancreatitis from that secondary to carcinoma often cannot be done solely on the basis of CT scanning. The CT scan in chronic pancreatitis may be normal, demonstrate calcific deposits within the pancreas, show an enlarged pancreatic duct, or reveal the pancreas to be small and atrophic. Pancreatic pseudocysts are readily detected by CT scanning due to the marked difference in the attenuation coefficients of pseudocysts and normal pancreatic tissue. The scan in pancreatic abscess characteristically shows an area of pancreatic enlargement that contains multiple ill-defined areas of low attenuation number. Disease within the peripancreatic lymphatic tissue produces a mass on CT scan that may be impossible to distinguish from pancreatic carcinoma. In some instances, however, the fat plane between the aorta, the mass, and the pancreas may be preserved and allow a preoperative diagnosis of peripancreatic lymphoma. CT scanning of the pancreas is a safe, rapid, and effective method of imaging the normal or abnormal pancreas of most patients who have a normal amount of body fat.

8789 CT SCANNING AND ULTRASOUND IN THE EVALUATION OF PANCREATIC PSEUDOCYSTS: A PRELIMINARY COMPARISON. (Eng.) Kressel, H. Y.; Mar-

gulis, A. R.; Gooding, G. W.; Filly, R. A.; Moss, A. A.; Korobkin, M. (Univ. California Sch. Medicine, San Francisco, CA 94143). *Radiology* 126(1):153-157; 1978.

A retrospective study of 12 surgically proved cases of pancreatic pseudocysts was carried out to assess computed tomography and ultrasound in the diagnosis of these lesions. Computed tomography correctly demonstrated six of eight uncomplicated pseudocysts and all of four infected pseudocysts. Ultrasound identified seven of the eight uncomplicated pseudocysts and two of the four infected pseudocysts. In the two remaining patients with infected pseudocyts, extensive bowel gas precluded optimal evaluation, and the limited examination failed to reveal evidence of an abnormal pancreatic fluid collection. One case of uncomplicated pseudocyst was missed by both tomography and ultrasound, but dilatation of the intrahepatic and common bile duct was noted. In the other case missed by tomography, scans were suboptimal in a markedly cachectic patient. The results indicate that errors by one modality are due to limitations not shared by the other method. This suggests a basis for the complementary use of computed tomography scanning and ultrasound in the evaluation of suspected pancreatic pseudocysts.

8790 STUDY OF PURE HUMAN PANCREATIC JUICE COLLECTED AFTER SECRETIN AND CERULEIN STIMULATION. (Fre.) Escourrou, J.; Frexinos, J.; Ribet, A. (Clinique des Maladies de l'Appareil Digestif, C.H.U. de Rangueil, F 31054 Toulouse, France). *Gastroenterol. Clin. Biol.* 2(1):29-37; 1978.

Pancreatic function after stimulation by simultaneous continuous infusion of 0.5 U/kg body weight/hr of secretin and 75 ng/kg body weight/hr of cerulein was studied in seven normal men. The patients had fasted for 36 hr and had no premedication except 10-20 mg *N*-butylhyoscine bromide used to produce duodenal hypotony when the catheter for collecting the pancreatic juice was introduced into the main pancreatic duct. The subjects were in the left-side position during the entire examination (60 min) to avoid contamination with gastric secretion. Samples were collected every minute for the first 15 min after injection of secretin and cerulein; thereafter, they were collected every 5 min. There was usually a latent period of 5 min before the pancreatic juice samples actually could be collected, which was attributed to the time the sample took to pass through the collecting catheter. The total collected volume varied from 118-210.7 ml and appeared related to the weight of the subject. The pancreatic juice pH was between 8.2-9.2. Bicarbonates rose to a maximum, varying from 116-140 mEq/l, at about 7-14 min. The sum of the bicarbonates and chlorides remained constant throughout the test for each subject and varied from 145.5 ± 1.7 mEq/l to 153.8 ± 0.9 mEq/l. Proteins, lipase, chymotrypsin, and amylase levels were also measured. The proportions of the different enzymes varied from one subject to another. It is concluded that collection of pancreatic fluid from the main pancreatic duct avoids contamination by bile or gastric juice with minimal danger to the subject.

8791 ^{99m}Tc-LABELED COMPOUNDS IN HEPATOBILIARY INVESTIGATIONS, AS EXEMPLIFIED BY STUDIES WITH HepatoBIDA. (Ger.) Wolf, F.; Kronert, E.; Regler, G. (Institut und Poliklinik fur Nuklearmedizin, Universitat Erlangen-Nurnberg, Krankenhausstrasse 12, 8520 Erlangen, W. Germany). *Fortschr. Med.* 96(9):455-462; 1978.

The pharmacokinetics and diagnostic usefulness of ^{99m}Tc-labeled 2,6-diethylacetanilidoiminodiacetic acid (HepatoBIDA) were studied in normal subjects and 52 patients with hepatobiliary disorders (mechanical obstruction of bile ducts, gallbladder and bile duct inflammation, dyskinesia, hepatic parenchymal lesion, liver metastasis, hydatid cyst, and hepatocellular carcinoma). HepatoBIDA was taken up by the liver very rapidly following the i.v. injection of 2-5 mCi. The plasma clearance was 420 ml/min after 10 min, and 250 ml/min after 30 min (body surface 1.73 m^2). The experimental studies on the patients showed that it is mandatory to obtain visual documentation of the marker passage by sequential scintigrams and to get functional graphs, especially from the complete examination field, the peripheral and central liver regions, the bile transporting system, and the small intestine. At present, the main indications for this examination are in the diagnosis of bile flow obstruction of mechanical or inflammatory origin and in the evaluation of therapeutic measures.

8792 VISUALIZATION OF THE LIVER, BILIARY TREE AND PANCREAS. PART II: RADIONUCLIDE EVALUATION. (Eng.) Spencer, R. P. (Univ. Connecticut Health Center, Farmington, CT). *Clin. Gastroenterol.* 7(2):475-487; 1978.

8793 VISUALIZATION OF THE LIVER, BILIARY TREE AND PANCREAS. PART III: ULTRASOUND SCANNING. (Eng.) Taylor, K. J. W.; Rosenfield, A. T. (Yale Univ. Sch. Medicine, New Haven, CT). *Clin. Gastroenterol.* 7(2):488-516; 1978.

8794 EXCRETORY LIVER FUNCTION TESTS. (Eng.) Bircher, J.; Preisig, R. (Inselspital Berne, Berne, Switzerland). *Clin. Gastroenterol.* 7(2):517-528; 1978.

8795 TESTS OF PANCREATIC SECRETION. (Eng.) Wormsley, K. G. (Dept. Therapeutics, Univ. Dundee, Dundee, Scotland). *Clin. Gastroenterol.* 7(2):529-544; 1978.

8796 BILE SALT DECONJUGATION BREATH TESTS AND THE COLONIC FLORA [Letter to Editor].

(Eng.) Gilat, T.; Levy-Gigy, C.; Peled, Y. (Ichilov Hosp., Tel-Aviv, Israel). *Gastroenterology* 74(2, Part 1):332-334; 1978.

8797 INNOMINATE GROOVES OF THE COLON: MORPHOLOGICAL CHARACTERISTICS AND ETIOLOGIC MECHANISMS. (Eng.) Cole, F. M. (McMaster Univ. Medical Center, Hamilton, Ontario L8S 4J9, Canada). *Radiology* 128(1):41-43; 1978.

8798 COMPARISON OF JUICE OBTAINED DURING DUODENAL ASPIRATION AND CANNULATION OF THE MAIN PANCREATIC DUCT AFTER STIMULATION WITH EXOGENOUS SECRETIN IN MAN. (Eng.) Osnes, M.; Petersen, H.; Schrumpf, E. (Ulleval Hosp., Oslo 1, Norway). *Scand. J. Gastroenterol.* 13(4): 453-458; 1978.

8799 A RADIOTELEMETRY PILL FOR THE MEASUREMENT OF IONISING RADIATION USING A MERCURIC IODIDE DETECTOR. (Eng.) Hassan, M. A.; Pearce, G.; Edwards, J. P. N. (Univ. Jordon, Amman, Jordon). *Phys. Med. Biol.* 23(2):302-308; 1978.

8800 THE VALUE OF PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY IN THE DIAGNOSIS OF DISEASES OF THE BILIARY TRACT. (Eng.) Middendorp, U. G.; Bessler, W.; Gasser, U. (Kantonsspital, Winterthur, Switzerland). *Chir. Gastroenterol.* 11(3):337-341; 1977.

8801 CHOLEDOCHOSCOPY: A COMPARISON OF A RIGID AND A FLEXIBLE FIBEROPTIC INSTRUMENT. (Eng.) Iseli, A.; Marshall, V. C. (Prince Henry's Hosp., St. Kilda Rd., Melbourne, Victoria 3004, Australia). *Med. J. Aust.* 1(3):131-132; 1978.

8802 THE FEASIBILITY OF ECHOMETRIC DETECTION OF CANCER IN THE LOWER GASTROINTESTINAL TRACT. (Eng.) Wild, J. J.; Foderick, J. W. (Univ. Cambridge, Cambridge, England). *Am. J. Proctol.* 29(1):16-25; 1978.

8803 THE FEASIBILITY OF ECHOMETRIC DETECTION OF CANCER IN THE LOWER GASTROINTESTINAL TRACT: PART II. (Eng.) Wild, J. J.; Foderick, J. W. (Univ. Cambridge, Cambridge, England). *Am. J. Proctol.* 29(2):11-20; 1978.

8804 DIAGNOSTIC TESTS OF EXOCRINE PANCREATIC FUNCTION AND DISEASE. (Eng.) Arvanitakis, C.; Cooke, A. R. (Kansas Univ. Medical Center, Rainbow Blvd. at 39th St., Kansas City, KS 66103). *Gastroenterology* 74(5, Part 1):932-948; 1978.

8805 METOCLOPRAMIDE IN GASTROINTESTINAL RADIOLOGY. (Eng.) Ho, C. S.; Rubin, E.; Renouf, J. H. P. (Toronto General Hosp., 101 College St., Toronto, Ontario M5G 1L7, Canada). *J. Can. Assoc. Radiol.* 29(1):51-55; 1978.

8806 ALTERED BODY DISTRIBUTION OF [^{99m}Tc]PERTECHNETATE IN LATROGENIC [sic] HYPERALUMINEMIA. (Eng.) Wang, T. S. T.; Fawwaz, R. A.; Esser, P. D.; Johnson, P. M. (Div. Nuclear Medicine, Columbia Univ., 630 W. 168th St., New York, NY 10032). *J. Nucl. Med.* 19(4):381-383; 1978.

8807 EARLY FIBEROPTIC PANENDOSCOPY IN ACUTE UPPER GASTROINTESTINAL BLEEDING. (Eng.) Mann, N. S.; Mann, S. K. (Veterans Admin. Hosp., 800 Zorn Ave., Louisville, KY 40202). *Am. J. Proctol.* 29(2):26-29, 31; 1978.

8808 EVALUATION OF VARIOUS DIAGNOSTIC TECHNIQUES FOR THE STUDY OF FAECAL PARASITES. (Eng.) Chugh, T. D.; Singh, J. (Medical Coll., Rohtak, India). *Indian Med. Assoc.* 69(11):245-247; 1977.

8809 LP-X IN MATURE AND PREMATURE NEWBORN INFANTS. (Eng.) Lachmann, D.; Eder, G. *In: Protides of the Biological Fluids. Proceedings of the Twenty-Fifth Colloquim, Brugge, 1977.* (Oxford: Pergamon Press): 337-340, 1977.

8810 ONE-HOUR BLOOD XYLOSE TEST: A RELIABLE INDEX OF SMALL BOWEL FUNCTION. (Eng.) Buts, J. P.; Morin, C. L.; Roy, C. C.; Weber, A.; Bonin, A. (Hopital Sainte-Justine, 3175 Chemin de la Cote Ste-Catherine, Montreal, Quebec H3T 1C5, Canada). *J. Pediatr.* 92(5):729-733; 1978.

8811 USE OF THE ONE-HOUR BLOOD XYLOSE TEST AS AN INDICATOR OF SMALL BOWEL MUCOSAL DISEASE. (Eng.) Christie, D. L. (Mason Clinic, 1100 Ninth Ave., Seattle, WA 98101). *J. Pediatr.* 92(5): 725-728; 1978.

8812 A RADIOLOGICAL EVALUATION OF SMALL BOWEL ACTIVITY IN THE ACUTE ABDOMEN. (Eng.) Bryk, D. (Jewish Hosp. and Medical Center, Brooklyn, NY). *C.R.C. Crit. Rev. Diagn. Imaging* 10(2):99-128; 1977.

8813 ULTRASONIC EXAMINATION OF THE GALLBLADDER: A REVIEW. (Eng.) Gonzalez, A. C.; Johnson, J. A., III (Emory Univ. Sch. Medicine, 80 Butler St., S.E., Atlanta, GA 30303). *Clin. Radiol.* 29(2):171-176; 1978.

8814 EXPERIENCE WITH ROUTINE OPEN ABDOMINAL PARACENTESIS. (Eng.) Manganaro, A. J.; Pachter, H. L.; Spencer, F. C. (New York Univ. Medical Center, New York, NY). *Surg. Gynecol. Obstet.* 146(5):795-796; 1978.

8815 THE BILIARY TRACT SYSTEM--A CHALLENGE TO DIAGNOSTIC RADIOLOGY. (Eng.) Frommhold, W. (Medizinischen Strahleninstituts der Universitat Tubingen, Roentgenweg, Tubingen 74, W. Germany). *J. Belge. Radiol.* 60(5):411-422; 1977.

8816 ENDOSCOPIC RETROGRADE CHOLANGIOGRAPHY USING BARIUM SULFATE: AN ALTERNATIVE APPROACH TO THE CONTRAST MEDIA-REACTIVE PATIENT. (Eng.) Severance, S. R.; Dagradi, A. E. (Veterans Admin. Hosp., 5901 E. Seventh St., Long Beach, CA 90822). *Am. J. Gastroenterol.* 69(1):57-62; 1978.

8817 STRANGULATING OBSTRUCTION OF THE BOWEL: A REEVALUATION OF RADIOGRAPHIC CRITERIA. (Eng.) Bryk, D. (State Univ. New York Downstate Medical Center, Brooklyn, NY). *Am. J. Roentgenol.* 130(5):835-843; 1978.

8818 CLINICAL EXPERIENCE WITH [^{111}In]INDIUM CHLORIDE SCANNING IN INFLAMMATORY DISEASES. (Eng.) Dayem, H. M. A.; Breen, J.; Leslie, E. V. (Dept. Radiology, State Univ. New York at Buffalo, 462 Grider St., Buffalo, NY 14214). *Clin. Nucl. Med.* 3(5):196-201; 1978.

8819 SCANTY INTESTINAL GAS IN INFANTS AND CHILDREN: CLINICAL SIGNIFICANCE AND RADIOLOGIC DIAGNOSIS. (Eng.) Eklof, O.; Lette, E. (Karolinska Sjukhuset, S-104 01 Stockholm, Sweden). *Acta Radiol.* [*Diagn.*] *(Stock.)* 19(13): 259-267; 1978.

8820 CLINICAL ESTIMATION OF LIVER SPAN IN INFANTS AND CHILDREN. (Eng.) Lawson, E. E.; Grand, R. J.; Neff, R. K.; Cohen, L. F. (Children's Hosp. Medical Center, 300 Longwood Ave., Boston, MA 02115). *Am. J. Dis. Child.* 132 (5):474-476; 1978.

8821 VALUE OF FIBEROPTIC GASTROINTESTINAL ENDOSCOPY IN INFANTS AND CHILDREN. (Eng.) Graham, D. Y.; Klish, W. J.; Ferry, G. D.; Sabel, J. S. (Baylor Coll. Medicine, 1200 Moursund Ave., Houston, TX 77030). *South. Med. J.* 71(5): 558-560; 1978.

8822 A SIMPLE ASSAY TECHNIQUE FOR PANCREATIC LIPASE. (Eng.) Connon, R. E.; Van Kley, H.; Knight, W. A., Jr. (St. Mary's Health Center, 6420 Clayton Rd., St. Louis, MO 63117). *Am. J. Dig. Dis.* 23(5):472-475; 1978.

8823 RADIOLOGIC VISUALIZATION OF THE NORMAL DUODENAL MINOR PAPILLA. (Eng.) Op den Orth, J. O. (St. Elisabeth's of Groote Gasthuis, Haarlem, Netherlands). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 128(5):572-576; 1978.

See also, 8744, 8774, 8775, 8813, 8836, 8873, 8890, 8901, 8902, 8906, 8926, 8927, 8934, 8935, 8937, 8938, 8953, 8960, 8962, 8963, 8964, 8965, 8971, 8975, 9000, 9001, 9025, 9028, 9038, 9041, 9042, 9043, 9046, 9048, 9050, 9063, 9072, 9083, 9087, 9092, 9094, 9100, 9105, 9110, 9114, 9157, 9184, 9198, 9200, 9201, 9203, 9206, 9207, 9210, 9260, 9262.

GASTROINTESTINAL DISEASES

ESOPHAGUS

8824 ENDOSCOPY IN LYE BURNS OF OESOPHAGUS AND STOMACH. (Eng.) Poelman, J. R.; Hausman, R. H.; Hoitsma, H. F. W. (Academic Hosp., Free Univ., De Boelelaan 1117, Amsterdam, Netherlands). *Endoscopy* 9(3):172-177; 1977.

The cases of two patients with caustic esophageal and gastric injuries following lye ingestion are presented. The patients were a 52-yr-old woman and a 67-yr-old man. Both presented with oral and pharyngeal pain, burning pain behind the sternum and upper abdomen, and hematemesis. Endoscopic examination was omitted in the first patient because of the danger of perforation. About 12 hr after admission, the patient went into hypovolemic shock, and signs of peritonitis developed. At laparotomy, total resection of the stomach, spleen, and gallbladder was performed because of massive necrosis. A perforation was not found, and it was assumed that the caustic solution had leaked through the stomach causing chemical peritonitis. A pancreatic fistula and subphrenic abscess developed during the postoperative period. Endoscopy was carried out in the second patient 6 hr after admission. Severe erosive, hemorrhagic, fibrinous esophagitis; gastric thrombosis of the superficial vessels with hyperemia, edema, erosions, necrotic spots, and hemorrhage; and pyloric atony, hyperemia, and edema were found. The duodenal mucosa was normal. The patient was treated conservatively, and the healing process was followed by repeat endoscopy. Stenosis did not occur. The authors conclude that the development of the pancreatic fistula and subphrenic abscess in the first patient might have been averted had endoscopy been performed.

8825 ESOPHAGEAL DYSPHAGIA TREATED BY CRICOPHARYNGEAL MYOTOMY. (Eng.) West, E. M., Jr.; Baker, H. W. (Good Samaritan Hosp., Portland, OR 97201). *Am. Surg.* 43(11):703-708; 1977.

The results of cricopharyngeal myotomy in the treatment of 14 patients with dysphagia were analyzed, and the technique is reviewed. Surgery is carried out via an oblique incision over the anterior border of the left sternocleidomastoid muscle in its lower one-half. The pharyngeal constrictors are identified and the cricopharyngeus is differentiated by the transverse fibers without a midline raphe, and the fibers are divided vertically with sharp dissection down to the esophageal mucosa. The incised fibers include the inferior fibers of the inferior pharyngeal constrictors as well as the superior fibers of the circular esophageal constrictors to insure complete division of the sphincter. This may also be facilitated by the use of a Foley catheter passed, then inflated and pulled up by the anesthesiologist. If the myotomy is complete, the esophageal mucosa should bulge through the myotomy site. A small Penrose drain is placed in the retroesophageal space prior to closure of the incision. The patients ranged in age from 46 to 86 yr. Nine were roentgenographically proven to have cricopharyngeal dysfunction. Seven patients with cricopharyngeal hypertrophy without other evident current disease had excellent results from myotomy, including one with remote bulbar poliomyelitis. One patient each with Parkinson's disease, hypopharyngeal carcinoma, amyotrophic lateral sclerosis, and cerebrovascular accident had poor results. There were two complications. One patient had a postoperative hemorrhage that required reexploration and drainage of a hematoma. The other, an elderly patient with a cerebrovascular accident, died of aspiration pneumonitis postoperatively. The accurate diagnosis and treatment of dysphagia depends upon adequate history, examination, and appropriate testing. X-ray evidence, particularly cine esophagram, is the diagnostic medium of choice in cricopharyngeal dysfunction and hypertrophy. Section of this muscle can bring immediate relief of dysphagia in patients with otherwise intact swallowing mechanisms.

8826 EPITHELIAL DYSPLASIA IN CANCEROUS AND NONCANCEROUS ESOPHAGI. (Eng.) Sato, E.; Tokunaga, M.; Sakae, K.; Mukada, T.; Sasano, N. (Kagoshima Univ. Faculty Medicine, Kagoshima 890, Japan). *Tohoku J. Exp. Med.* 124(2):117-128; 1978.

To examine the role of epithelial dysplasia (ED) in carcinogenesis of the esophagus, the incidence of ED in 86 autopsy samples of noncancerous esophagi and its degree in 100 surgical samples of esophageal carcinoma were determined by subserial examination of the autopsy samples and by semiserial examination of the surgical samples. The prevalence, severity, distribution, and histological features of dysplasia were examined in relation to tobacco and alcohol intake in the autopsy cases and in relation to preoperative treatment in the surgical cases. Some degree of dysplasia was found in 55 of the autopsy cases. The higher grades of dysplasia were found in 17 men and in 3 women. Some correlation between the use of alcohol and cigarettes and the incithe use of alcohol and cigarettes and the incidence and severity of dysplasia was evident but not conclusive due to incomplete data. Extensive use of alcohol appeared to correlate with more severe lesions. A definite increase in incidence and severity of dysplasia occurred with increasing age. In 13 cases, atypical active elongation of the germinal layer simulating the rete ridge was observed. Chronic inflammation, glycogenic acanthosis and hyperplasia of duct epithelium were frequently noted. In 70 of 100 surgical samples, an intimate correlation of carcinoma and dysplasia was observed, the degree of dysplasia being generally more advanced than in the autopsy samples. In 24 instances, the carcinoma was accompanied by extensive dysplasia and in 46 instances by multifocal dysplasia. Multicentric *in situ* carcinoma,

frequently surrounded by severe dysplasia, was found in 19 patients. The lesion of invasive carcinoma sometimes did not show sharp demarcation from the surrounding mucosa. The effect of radiation or radiation plus bleomycin therapy on carcinoma tissue is noted. The autopsy data on the higher incidence of higher grades of dysplasia among men correlated with the documented 3.84:1 male/female ratio of death from esophageal cancer in the region. The studies on surgical samples, particularly those with no preoperative therapy, indicate that at least half of the carcinoma of the esophagus arose from the dysplastic epithelium as a precursor lesion.

8827 FIBERENDOSCOPIC MONITORIZED DILATATION OF OESOPHAGEAL STRICTURES IN CHILDREN. (Eng.) Cadranel, S.; Rodesch, P.; Peeters, J. P.; Cremer, M.; Cremer, N. (Dept. Pediatrics, Univ. Brussels, Brussels, Belgium). *Endoscopy* 9(3):127-130; 1977.

Experience with esophageal dilatation in 10 children aged 2 months to 10 yr with acquired esophageal strictures is presented. Causes included peptic stricture (3 cases), lye ingestion (2 cases), radionecrosis (1), extrinsic compression (1), congenital atresia (2), and granuloma (1). There were a total of 55 dilatations, using the classical method with rubber bougies in three patients and a newer technique using metallic olives in the remaining patients. There was one case of perforation, which occurred in a patient with caustic stricture in whom dilatation was accomplished with rubber bougies. The only other complication was laceration, occurring in a patient with congenital atresia who was treated with the metallic olives. The results obtained were judged to be excellent in one case in which metallic olives were used and good in the remainder of these cases. Good results were obtained with the classical method in the two cases without perforation. Relapses occurred in only two patients, both of whom underwent bougienage with metallic olives: in one patient with caustic stricture and in one patient with initial caustic stricture followed by relapse with peptic stricture.

8828 SIMPLIFIED OESOPHAGEAL TRANSECTION FOR BLEEDING VARICES. (Eng.) Johnston, G. W. (Royal Victoria Hosp., Belfast, Northern Ireland). *Br. Med. J.* 1(6124):1388-1391; 1978.

8829 EOSINOPHILIC ESOPHAGITIS IN A PATIENT WITH VIGOROUS ACHALASIA. (Eng.) Landres, R. T.; Kuster, G. G. R.; Strum, W. B. (Scripps Clinic Medical Inst., La Jolla, CA). *Gastroenterology* 74(6):1298-1301; 1978.

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8831 MALLORY-WEISS SYNDROME WITH ESOPHAGEAL OBSTRUCTION SECONDARY TO ADHERENT INTRALUMINAL THROMBUS. (Eng.) Curtin, M. J.; Milligan, F. D. (St. Mary's Hosp. Medical Center, 707 S. Mills St., Madison, WI 53715). *Am. J. Roentgenol.* 129(3):508-510; 1977.

8832 TREATMENT OF BLEEDING VARICES BY OESOPHAGEAL TRANSECTION WITH THE SPTU GUN. (Eng.) Johnston, G. W. (Royal Victoria Hosp., Belfast, Northern Ireland). *Ann. R. Coll. Surg. Engl.* 59(5):404-408; 1977.

8833 CARCINOMA OF THE ESOPHAGUS: A NEW PROPOSAL FOR THE EVALUATION OF TREATMENT. (Eng.) Stoller, J. L.; Samer, K. J.; Toppin, D. I.; Flores, A. D. (Laurel Medical Center, 316-888 W. 8th Ave., Vancouver, British Columbia V5Z 3Y1, Canada). *Can. J. Surg.* 20(5):454-459; 1977.

8834 DOXYCYCLINE ESOPHAGEAL ULCERS. (Eng.) Schneider, R. (St. Thomas Hosp., P.O. Box 380, Nashville, TN 37202). *Am. J. Dig. Dis.* 22(9):805-807; 1977.

8835 DISSEMINATED HISTOPLASMOSIS PRESENTING WITH ESOPHAGEAL SYMPTOMATOLOGY. (Eng.) Lee, J. H.; Neumann, D. A.; Welsh, J. D. (Univ. Oklahoma Health Sciences Center, P.O. Box 26901, Oklahoma City, OK 73190). *Am. J. Dig. Dis.* 22(9): 831-834; 1977.

8836 FLUORESCENCE TECHNIQUE FOR THE VERIFICATION OF VASCULARISATION IN PEDICULATED SEGMENTS OF THE DIGESTIVE TRACT. (Eng.) Garcia Gutierrez, A.; Mederos Pazos, G.; Roque Zambrana, F.; Garcia Funes, O. (Garcia's Hosp., Havana, Cuba). *Chir. Gastroenterol.* 11(2):193-196; 1977.

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8838 NISSEN FUNDOPLICATION FOR REFLUX PEPTIC ESOPHAGITIS. (Eng.) Bushkin, F. L.; Neustein, C. L.; Parker, T. H.; Woodward, E. R. *In: Transactions of the Southern Surgical Association Eighty-Eighth Annual Meeting held at The Breakers, Palm Beach, Florida, December 6, 7, 8, 1976.* (Philadelphia: J. B. Lippincott): 88:180-185; 1977.

8839 DIFFUSE ESOPHAGEAL SPASM PROVOKED WITH PROSTIGMINE [Letter to Editor]. (Eng.)

DeJesus, R.; Peternel, W. W.; Orchard, J. L.; Colatrella, A. M. (Mercy Hosp., 1400 Locust St., Pittsburgh, PA 15219). *Gastroenterology* 74(2, Part 1):332; 1978.

8840 EXPERIENCES IN THE TREATMENT OF ESOPHAGEAL CANCER OF THE UPPER AND MIDDLE THORACIC SEGMENT. (Eng.) Nakayama, K. (Nakayama Cancer Res. Inst., Tokyo, Japan). *Surg. Annu.* 9:125-132; 1977.

8841 THE COLUMNAR-LINED (BARRETT'S) ESOPHAGUS. (Eng.) Bremner, C. G. (Dept. Surgery, Univ. Witwatersrand, Johannesburg, South Africa). *Surg. Annu.* 9:103-123; 1977.

8842 THE SIGNIFICANCE OF ABNORMAL DUODENAL LOOP ON RADIOLOGICAL AND CLINICAL CURE IN INFANTS WITH SLIDING HIATAL HERNIA. (Eng.) Thommesen, P.; Gravesen, M.; Garsdal, L. (Municipal Hosp., DK-8000 Aarhus, Denmark). *Pediatr. Radiol.* 6(2):88-91; 1977.

See also, 8807, 8821, 8851, 8860, 8862, 8866, 8886, 8887, 8896, 8907, 9163, 9164, 9231, 9242.

STOMACH

8843 GASTRIC LESIONS IN FAMILIAL ADENOMATOSIS COLI: THEIR INCIDENCE AND HISTOLOGIC ANALYSIS. (Eng.) Watanabe, H.; Enjoji, M.; Yao, T.; Ohsato, K. (Faculty Medicine, Kyushu Univ. 60, 3-1-1, Maidashi, Higashi-Ku, Fukuoka 812, Japan). *Hum. Pathol.* 9(3):269-283; 1978.

A histologic analysis of the gastric lesions accompanying familial adenomatosis coli and an evaluation of the differences between polyps in the gastric antrum and those elsewhere in the body was conducted by x-ray and endoscopy of the stomachs of 22 patients with familial adenomatosis of the colon and rectum in 14 families. Although 7 patients did not have gastric lesions, in 15 of the patients (from 13 families), one or more types of gastric lesion was found. Gastric lesions were found in all 9 patients above the age of 30 yr and in 6 of 11 patients aged 10-29 yr. The lesions (1 in 3 cases, 3 in 1 case, 4 in 1 case, 5 in 1 case, and >5 in 9 cases) were limited to the antrum in eight cases, found elsewhere in the body in five, and present in both the antrum and other sites in two. The most common gastric lesion was the adenoma occurring in the pyloric mucosa, which was found in nine patients. Multiple lesions (maximum number, 43) were found in five cases. While there were no essential histologic differences between the gastric adenomas in the patients with familial adenomatosis coli and those in patients without the disease, the adenomas appeared at younger ages in patients with the disease and were multiple in 55.6% of these cases (versus 21.4% in patients without the disease). Fundic gland polyposis was the second most common gastric lesion in patients with familial adenomatosis coli, occurring in 6 of the 15 patients. The multiple fundic gland polyps, measuring up to 5 mm and limited to the fundic glandular mucosa, were characterized histologically by simple hyperplasia of the fundic glands. Other lesions detected included multiple gastric carcinoma (3), microcarcinoids in the pyloric mucosa (2), and multiple varioliform lesions representing erosive gastritis (2). The density of colonic adenomas was determined. Gastric lesions were found in all nine cases in the high density (innumerable adenomas) group, in four of six cases with a moderate number (500-2,000) of adenomas, and in only two of seven cases with low density of adenomas (50-200). Furthermore, extra-alimentary lesions were found in 9 of the 15 adenomatosis coli patients with gastric lesions but were present in only 1 of 7 patients without them. It is concluded that familial adenomatosis coli, Gardner's syndrome, and Turcot's syndrome are all expressions of the familial adenomatosis coli syndrome. Fundic gland polyposis seems to be the gastric lesion specific to familial adenomatosis of the colon and rectum.

8844 HUMAN NEWBORN HYPERGASTRINEMIA: AN INVESTIGATION OF PRENATAL AND PERINATAL FACTORS AND THEIR EFFECTS ON GASTRIN. (Eng.) Euler, A. R.; Ament, M. E.; Walsh, J. H. (Univ. California, Sch. Medicine, Los Angeles, CA). *Pediatr. Res.* 12(5):652-654; 1978.

Levels of gastrin secretion in newborns and adults were compared and the effect of a variety of prenatal and perinatal conditions on these levels in newborns was investigated. Serum gastrin values were determined by radioimmunoassay in 217 newborn infants and in 802 adults who had peptic ulcer disease symptoms but did not have Zollinger-Ellison syndrome or peptic ulcer disease. In six newborns, the basal acid secretion during the first 8 hr of life was measured and compared with that of 14 children with functional abdominal pain. Newborns had a median gastrin serum of 100 pg/ml while the adult median was 39 pg/ml. Mean values for newborns and adults were 135 and 49 pg/ml, resp., ($p<0.001$). No adult had gastrin levels greater

than 200 pg/ml, but 29 newborns had gastrin determinations greater than 200 pg/ml, and 5 had values above 500 pg/ml. None of the 19 maternal medications studied, including oxytocin and atropine, were found to influence the newborn gastrin levels. Of 35 neonatal and maternal factors analyzed, only maternal sickle cell trait and race were shown to influence these levels. Only two mothers in the study had sickle cell trait, and their infants had gastrin levels of 1,000 and 270 pg/ml. Negro newborns had a significantly higher level of gastrin than did either Caucasian or Chicano infants, the mean log gastrin values (2.16%, 2.029%, and 1.97%, resp.) corresponding to serum levels of 147, 93, and 107 pg/ml (F=4.7, significant at 5% level). Serum gastrin levels continued to be elevated through the first 8 hr of life (mean value at 8 hr, 72 pg/ml). The basal acid secretion study demonstrated that the parietal cells of newborns secrete only small amounts of gastric acid in response to the elevated gastrin levels. Compared to the older children, the infants hyposecrete acid in response to gastrin stimulus.

8845 GASTRIC MUCOSAL CELL PROLIFERATION AND IMMUNOGLOBULIN-CONTAINING CELLS IN MENETRIER'S DISEASE. (Eng.) Hansen, O. H.; Jensen, K. B.; Larsen, J. K.; Soltoft, J. (Bispebjerg Hosp., DK-2400 Copenhagen NV, Denmark). *Digestion* 16(4): 293-298; 1977.

The amount of immunoglobulin-containing cells in the gastric mucosa of six patients with Menetrier's disease was estimated, and the rate of gastric epithelial cell proliferation was studied in four of these patients. The area of cells containing immunoglobulin in the gastric fundic mucosa was calculated as a percentage of the total interstitial area in the lamina propria. The areas of IgA- and IgG-containing cells were within normal limits, but the area of IgM-containing cells was significantly increased (mean 9.5% compared with the normal 2.4 ± 1.8%). In three biopsies from the antrum, the area of IgM-containing cells was increased in only one. Autoradiographic studies in four patients showed that the mean labeling indices in the antral and fundic mucosa (15.2 ± 1.4 and 13.3 ± 2.3, resp.) were not significantly different from those previously reported in patients without gastric disease. The serum IgM levels were normal in all six patients. The results indicate that the number of IgM-containing cells in gastric fundic mucosa is markedly increased. This may indicate that IgM is involved in the pathogenesis of Menetrier's disease or that the increased number of IgM-containing cells is the result of a continuous antigen challenge across the much enlarged epithelial membrane.

8846 FACTORS RELEVANT TO THE OCCURRENCE, RECURRENCE AND HEALING OF CHRONIC GASTRIC ULCER: A STUDY OF AN AUSTRALIAN POPULATION. (Eng.) Thomas, J.; Greig, M.; Shinners, J.; Piper, D. W. (North Shore Hosp., St. Leonards, 2065, New South Wales, Australia). *Rend. Gastroenterol.* 9(2):109-113; 1977.

To evaluate factors relevant to the development, recurrence, and healing of chronic gastric ulcers, 216 patients with ulcer were compared with controls matched for age, sex, and social grade. Emotional stress was measured by the frequency of 31 "major life events" in 59 patients and controls. Patients in the lower social grades experienced more of these events and had a significantly higher score for the degree of change that these events created in the patients' lives ($p<0.05$) than did the controls. The personality of 44 patients with ulcer was compared with that of controls, using the self-rating Eysenck Personality Inventory. Men with chronic gastric ulcer were significiantly more neurotic ($p<0.01$) and less extroverted ($p<0.001$) than their controls. Forty-three percent of 49 patients with recurrent ulcer had a positive test for urinary salicylates, compared with 24% of their controls ($p<0.05$). There was no significant difference in the number of positive urinary salicylate tests among 22 patients in remission and their controls. More patients than controls lived alone ($p<0.01$). In addition, patients reported more tension in the home than did their controls, but the results failed to reach statistical significance. Previous studies linking cigarette smoking with slower ulcer healing or with a greater incidence of recurrence could not be supported in this series. Bed rest, carbenoxolone sodium treatment, and anticholinergic drug therapy also had no influence on ulcer healing in the 54 hospitalized patients studied. Cimetidine was found to be effective in a double-blind controlled trial of 48 inpatients and outpatients.

8847 CLINICAL STUDY ON GASTRIC SECRETION WITH SPECIAL REFERENCE TO PEPSIN SECRETION. (Eng.) Ito, K.; Inoue, M.; Noda, T.; Kuwano, K.; Takeda, H.; Kasugai, T.; *et al.* (Fukuoka Univ. Sch. Medicine, Fukuoka, Japan). *Gastroenterol. Jpn.* 13 (1):13-20; 1978.

Gastric acid and pepsin secretion were measured in groups of 10-24 patients with peptic ulcer to evaluate the preferential stimulating action of tetragastrin (4 µg/kg, i.m.), cerulein (0.2 or 0.3 µg/kg, i.m.), Histalog (1 and 2 mg/kg, i.m.), and secretin (1 clinical U/kg diluted with 15 ml of distilled water, i.v.). Tetragastrin produced a peak acid concentration and volume 30 min after administration. Acid concentration was significantly ($p<0.001$) increased over basal secretion. A significant ($p<0.05$) increase in peptic activity occurred 10 min after stimulation and then decreased gradually. Pepsin output increased 30 min after stimulation. Both doses of cerulein produced significant ($p<0.05$) increases in acid concentration. Increases in secretory volume and in peptic activity after cerulein injection were not significant compared with basal levels. Histalog produced a high level of peptic activity at 10 min after stimulation, which was maintained up to 30 min and was significantly ($p<0.05$) different from basal values. The acid concentration increased gradually, and the peak was observed 60 min after stimulation. At 60 min, the acid concentration was significantly

higher than basal values ($p<0.01$ for 1 mg/kg and $p<0.001$ for 2 mg/kg Histalog). After secretin injection, peptic activity increased significantly ($p<0.01$, peak at 40 min) and acid concentration decreased significantly ($p<0.02$, peak at 10-30 min after stimulation). With tetragastrin or cerulein as stimulant, the increase in peptic activity depended on the increase in acid secretion. Since only a slight increase in pepsin secretion was seen when cerulein was used, the authors conclude that cerulein stimulates parietal cells but will not stimulate chief cells. The increased peptic activity following the increase of acid concentration may be based on "wash-out" action caused by increase of acid secretion after stimulation. The secretory pattern observed when Histalog was the stimulant indicates that Histalog stimulates the chief cells independent of acid secretion. Secretin also stimulates chief cells. The results indicate that Histalog is a more adequate stimulant for pepsin secretion than is gastrin or cerulein.

8848 DOUBLE-BLIND STUDY OF CARBENOXOLONE IN GASTRIC ULCER AND EROSIONS. (Eng.) Lehtola, J.; Karvonen, A. L.; Tunturi-Hihnala, H. (Univ. Central Hosp., SF-90220 Oulu 22, Finland). *Ann. Clin. Res.* 10(1):19-23; 1978.

The value of carbenoxolone in the treatment of gastric ulcers and erosions was studied, and its effects on gastric acid secretion and on serum gastrin levels were investigated. In a double-blind study, 20 patients with chronic gastric ulcer and 20 patients with gastric erosions received either placebo or carbenoxolone sodium (50 mg) during 6 weeks. All patients also received antacids and were placed on an anti-ulcer diet. Gastroscopy was carried out at the onset of treatment and 3 and 6 weeks after the start of treatment. The ulcers of five patients receiving carbenoxolone healed during the 6-week treatment period, whereas ulcers in four patients in the placebo group healed during this period. Erosions in seven patients receiving carbenoxolone healed during the 6-week period, and erosions in 11 patients in the placebo group healed during this period; in two patients the erosions that had healed after 3 weeks' treatment recurred at 6 weeks. There were no significant differences at any time during the study in the healing of gastric ulcers or erosions between the patients receiving placebo and those receiving carbenoxolone. However, after 3 weeks' treatment, the patients receiving carbenoxolone were subjectively in better general condition than were the patients receiving placebo (difference statistically significant at the 5% level). No significant changes in the maximal acid output or in the serum gastrin levels were seen in the ulcer or erosion patients before and after carbenoxolone or placebo treatment. No cardiovascular side-effects were evident during carbenoxolone treatment, although one patient required potassium supplementation. The small dosage may explain the negligible effect of carbenoxolone on the healing of the gastric lesions and on the low incidence of side-effects in this study.

8849 EFFECT OF ULCER HEALING ON THE PROGNOSIS OF CHRONIC GASTRIC ULCER: FOUR-YEAR FOLLOW-UP. (Eng.) Piper, D. W.; Shinners, J.; Greig, M.; Thomas, J.; Waller, S. L. (Royal North Shore Hosp., Sydney, Australia). *Gut* 19(5):419-424; 1978.

To determine the influence of complete healing at the time of discharge from the hospital on the prognosis of chronic gastric ulcer, 83 patients were followed up for 4 yr after an admission to hospital with a chronic gastric ulcer proven by radiology (index ulcer). The index ulcer healed completely in 50 patients, but was unhealed at the time of discharge in the other 33. Significantly fewer patients whose index ulcer was healed compared with those whose ulcer was unhealed at discharge had had a recurrence by 1 ($p<0.05$), 2, 3, and 4 yr ($p<0.01$) of follow-up. Moreover, the rate of recurrence was significantly greater for those patients with an unhealed index ulcer compared with those with a healed index ulcer ($p<0.05$), and by the end of the 4-yr period, 61% of patients with unhealed compared with 26% of those with healed index ulcers had had a recurrence. Patients over the age of 60 yr with unhealed index ulcers had a significantly poorer prognosis than those of similar age whose index ulcer was healed at discharge ($p<0.025$). The sex of the ulcer patient did not significantly influence ulcer recurrence. Large ulcers (>57 mm^2) were less often healed on initial discharge and were more frequently associated with recurrence at 1 ($p<0.05$), 2 ($p<0.025$) 3, and 4 yr ($p<0.01$) than were smaller ulcers (≤57 mm^2). Heavy intake of analgesics adversely influenced the course of patients whose ulcer was unhealed ($p<0.05$), but did not alter the recurrence rate in those whose ulcer was healed. Ulcer recurrence was not influenced by smoking habits or by alcohol consumption.

8850 COMPOSITION OF THE ALKALINE COMPONENT OF HUMAN GASTRIC JUICE: EFFECT OF SWALLOWED SALIVA AND DUODENO-GASTRIC REFLUX. (Eng.) Okosdinossian, E. T.; El Munshid, H. A. (Dept. Physiology, Univ. Khartoum, P.O. Box 102, Khartoum, Sudan). *Scand. J. Gastroenterol.* 12(8):945-950; 1977.

To further substantiate the two-component hypothesis of gastric secretion (i.e., that gastric juice contains an acid and an alkaline component), the composition of alkaline gastric juice from four achlorhydric or hypochlorhydric patients with vitiligo and one patient with pernicious anemia was studied. Saliva swallowing was alternately allowed or prevented, and in this way the contribution of swallowed saliva to the pure alkaline component was assessed. The volume refluxed from the duodenum was estimated to assess the contribution of duodenal juice. Reflux was absent in one patient and negligible in another, while in the remaining three patients, the mean pyloric reflux amounted to no more than 8% of the observed volume. Swallowed saliva had a significant diluting effect on both the Na^+ and Cl^- concentrations ($p<0.01$ and $p<0.001$, resp.), but the K^+ concentration was significantly increased in the

alkaline gastric aspirate ($p<0.05$). The composition of alkaline gastric juice, free to an appreciable extent of salivary contamination, was relatively constant. The results are consistent with the two-component hypothesis of gastric secretion.

8851 GASTRIC VOLVULUS UNASSOCIATED WITH HIATAL HERNIA. (Eng.) Pillay, S. P.; Angorn, I. B.; Baker, L. W. (Dept. Surgery, Univ. Natal, Durban, South Africa). *S. Afr. Med. J.* 52(22): 880-885; 1977.

Clinical findings in 16 patients with gastric volvulus not associated with hiatal hernia are reported, and two case reports are presented. Of 10 patients who presented with volvulus unrelated to previous trauma, 3 presented with an acute volvulus, which constituted an abdominal emergency, with either obstruction or strangulation of the stomach, and required surgery. Of the six patients with relevant antecedent trauma, five presented with acute volvulus. Both deaths in this series were associated with an acute presentation. Two patients, who were both in the group with antecedent trauma, developed gangrene of part of the stomach wall, which resulted in the death of one patient before emergency surgery could be performed. All 16 patients presented with abdominal pain and vomiting, but a third common observation in gastric volvulus, i.e., inability to pass a nasogastric tube, was absent in all cases. Severe hematemesis due to gangrene or erosive gastritis occurred in three patients. Contrast studies of the upper gastrointestinal tract confirmed the diagnosis in 14 patients. Congenital diaphragmatic abnormalities were present as a causative factor in four patients. Diaphragmatic defects in the six patients with antecedent trauma were due to blunt or penetrating diaphragmatic injury, which preceded the admission for volvulus by intervals of 6 months to 4 yr. Alimentary tract lesions in the esophagus (1), stomach (2), and duodenum (1) were detected in four patients. In one patient, both a congenital defect of the hemidiaphragm and a gastric ulcer were present. Idiopathic volvulus was diagnosed in four cases. Among the patients without previous trauma, two with mesenterio-axial volvulus responded well to dietary modification. Plication of a congenital eventration of the left hemidiaphragm, gastropexy, and subphrenic transposition of the colon were associated with a good result in one patient, but volvulus recurred after gastropexy and colon transposition in a second patient, in whom a previously unrecognized gastric ulcer was discovered. The presence of an associated gastric ulcer was overlooked in another patient, in whom secondary gastric volvulus was presumed to be due to eventration of the left hemidiaphragm. In three patients with traumatic volvulus, thoracotomy exploration permitted easy reduction and the repair of diaphragmatic defects. Exploration via laparotomy in two patients was associated with great difficulty in dividing intrathoracic adhesions, reducing the volvulus and repairing the diaphragm. The rational management of gastric volvulus involves recognition that the condition may be idiopathic or secondary, treatment of associated conditions on their merits, awareness of diaphragmatic injury as a possible causative factor, and the use of gastropexy and subphrenic colonic displacement where indicated to prevent recurrence.

8852 GASTRITIS Varioliformis: CHRONIC EROSIVE GASTRITIS WITH PROTEIN-LOSING GASTROPATHY. (Eng.) Clarke, A. C.; Lee, S. P.; Nicholson, G. I. (Auckland Hosp., Park Rd., Auckland 3, New Zealand). *Am. J. Gastroenterol.* 68(6):599-602; 1977.

8853 MASSIVE UPPER GASTROINTESTINAL HEMORRHAGE FROM A GRANULAR CELL TUMOR OF THE STOMACH. (Eng.) Ross, J. S. (Berkshire Medical Center, Pittsfield, MA 01201). *Am. J. Gastroenterol.* 68(6):595-598; 1977.

8854 ACQUIRED DOUBLE PYLORUS. (Eng.) Hunt, R. H.; Day, R. C.; Jewell, D. P. (Royal Free Hosp., London NW3 2QG, England). *Br. Med. J.* 1(6115):759; 1978.

8855 CIMETIDINE IN THE MANAGEMENT OF GASTRIC ULCERATION. (Eng.) Colin-Jones, D. G. (Queen Alexandria Hosp., Portsmouth, England). *Scott. Med. J.* 22(4):287-297; 1977.

8856 THE USE OF DILUTE HYDROCHLORIC ACID AND CIMETIDINE TO REVERSE SEVERE METABOLIC ALKALOSIS. (Eng.) Rowlands, B. J.; Tindall, S. F.; Elliott, D. J. (Northern General Hosp., Sheffield S5 7AU, England). *Postgrad. Med. J.* 54(628): 118-123; 1978.

8857 GENETIC ASPECTS OF GASTRITIS-CANCER RELATIONSHIP. (Eng.) Varis, K.; Ihamaki, T.; Lehtola, J.; Sipponen, P.; Kekki, M.; Isokoski, M.; *et al.* (Meilathi Hosp., SF-Helsinki, Finland). *Dtsch. Z. Verdau Stoffwechselkr.* 38(2):51-54; 1978.

8858 AN EVALUATION OF GASTRIC CANCER SCREENING PROGRAM IN HISAYANA, JAPAN. (Eng.) Nakamura, Y.; Takeshita, M.; Hirota, Y.; Ueda, K.; Yao, T.; Kohzuki, T.; *et al.* (Fukuoka City Medical Association Hosp., 7-29, 2-Chome, Yakuin, Chuo-ku, Fukuoka 810, Japan). *Gastroenterol. Jpn.* 12(6):427-434; 1977.

8859 MISLEADING RESPONSE OF MALIGNANT GASTRIC ULCERS TO CIMETIDINE. (Eng.) Taylor, R. H.; Menzies-Gow, N.; Lovell, D.; La Brody, S. J.; Misiewicz, J. J. (Central Middlesex Hosp., London, England). *Lancet* 1(8066):686-688; 1978.

8860 ALIMENTARY TRACT SURGERY AND LYSOZYME ACTIVITY. (Eng.) Aoki, Y.; Sasaki, M.; Takei, N.; Yamada, Y.; Katsumi, M. (Wakayama Medical

Coll., Wakayama City, Wakayama 640, Japan). *Arch. Jpn. Chir.* 47(1):84-89; 1978.

8861 GASTRIC PHYTOBEZOARS CONSEQUENT TO DELAYED GASTRIC EMPTYING. (Eng.) Brady, P. G. (Univ. Kentucky Medical Center, Lexington, KY 40506). *Gastrointest. Endosc.* 24(4):159-161; 1978.

8862 INTRODUCTION OF A METHOD OF VALVULOPLASTIC ESOPHAGOGASTROSTOMY IN PROXIMAL GASTRECTOMY. (Eng.) Hatafuku, T.; Higuchi, T.; Seta, K. (Iwate Medical Univ. Sch. Medicine, Morioka 020, Japan). *Tohoku J. Exp. Med.* 124(2):129-138; 1978.

8863 POLYPOID GASTRITIS: REPORT OF A CASE ASSOCIATED WITH GASTRIC ADENOCARCINOMA AND REVIEW OF THE LITERATURE. (Eng.) Sanner, C. J.; Saltzman, D. A.; Mueller, J. C. (Univ. Arizona Medical Center, Tucson, AZ 85724). *Am. J. Dig. Dis.* 23(5, Suppl.):19s-24s; 1978.

8864 GASTRIC CANDIDIASIS. (Eng.) Gillespie, P. E.; Green, P. H. R.; Barrett, P. J.; Riley, J. W.; Nagy, G. S. (Royal North Shore Hosp., Sydney, Australia). *Med. J. Aust.* 1(4):228-229; 1978.

8865 A PROSPECTIVE COMPARATIVE STUDY OF CLINICAL AND PATHOLOGICAL CHARACTERISTICS IN ICELANDIC AND DANISH PATIENTS WITH GASTRIC ULCER, DUODENAL ULCER, AND X-RAY NEGATIVE DYSPEPSIA. II. HISTOLOGICAL RESULTS. (Eng.) Oddsson, E.; Binder, V.; Thorgeirsson, T.; Jonasson, T. A.; Gunnlaugsson, O.; Wulff, M.; *et al.* (St. Josefsspitalinn, Reykjavik, Iceland). *Scand. J. Gastroenterol.* 13(4):489-495; 1978.

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8867 ORAL DOMPERIDONE (R 33 812) IN THE TREATMENT OF CHRONIC DYSPEPSIA: A MULTICENTER EVALUATION. (Eng.) Lienard, J.; Janssen, J.; Verhaegen, H.; Bourgeois, E.; Willocx, R. (avenue du Polo, 60, B-1150 Brussels, Belgium). *Curr. Ther. Res.* 23(5):529-537; 1978.

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8869 TREATMENT OF GASTRIC ULCER: THE RANDOMIZED CLINICAL TRIALS FROM 1964 TO 1974 AND THEIR IMPACT. (Eng.) Christensen, E.; Juhl, E.; Tygstrup, N. (Rigshospitalet, Blegdamsvej 9, DK-2100 Copenhagen, Denmark). *Am. J. Gastroenterol.* 69(3):272-282; 1978.

8870 SPECIFIC CANCER IMMUNOTHERAPY ADJUNCTIVE TO SURGERY FOR ADVANCED GASTRIC CANCER: A REPORT OF SIX LONG TERM SURVIVORS. (Eng.) Ishikawa, Y.; Fukushima, M.; Machida, S.; Kakuta, H.; Nishikawa, T.; Fukuda, S. (Hirosaki Univ. Sch. Medicine, Zaifu-cho 5-chome, Hirosaki, 036 Japan). *Gastroenterol. Jpn.* 13(1):54-60; 1978.

8871 ENDOSCOPIC RESECTION OF AN OBSTRUCTING ANTRAL WEB IN AN INFANT. (Eng.) Brandt, L. J.; Boley, S. J.; Daum, F.; Kleinhaus, S. (Montefiore Hosp., 111 E. 210 St., Bronx, NY 10467). *Am. J. Dig. Dis.* 23(5, Suppl.):6s-12s; 1978.

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8873 ARTERIOVENOUS MALFORMATION OF THE STOMACH: RADIOLOGIC AND ENDOSCOPIC FEATURES. (Eng.) Lewis, T. D.; Laufer, I.; Goodacre, R. L. (McMaster Univ. Medical Center, Hamilton, Ontario L8S 4J9, Canada). *Am. J. Dig. Dis.* 23(5):467-471; 1978.

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PEPTIC ETIOLOGY? (Eng.) Gupta, A.; Hollander, D. (Wayne State Univ. Sch. Medicine, Gordon Scott Hall, 540 E. Canfield, Detroit, MI 48201). *Am. J. Dig. Dis.* 22(9):829-830; 1977.

8897 CONTROL OF GASTRIC ACIDITY BY GLYCOPYRROLATE PREMEDICATION IN THE PARTURIENT. (Eng.) Baraka, A.; Saab, M.; Salem, M. R.; Winnie, A. P. (Dept. Anesthesiology, American Univ. Beirut, Beirut, Lebanon). *Anesth. Analg. (Cleve.)* 56(5): 642-645; 1977.

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8901 THE DIAGNOSTIC VALUE OF X-RAY METHODS, ENDOSCOPY, ORIENTED GASTROBIOPSY AND BRUSH CYTODIAGNOSTICS IN MALIGNANT AND BENIGN DISEASES OF THE STOMACH. (Eng.) Brailski, C.; Malinova, B.; Popov, P. (Inst. Gastroenterology, Medical Academy, Belo More 8, Sofia, Bulgaria). *Arch. Geschwulstforsch.* 48(1):50-56; 1978.

See also, 8672, 8675, 8687, 8697, 8805, 8821, 8824, 8902, 8903, 8904, 8906, 8907, 8923, 8924, 8992, 9006, 9009, 9028, 9030, 9164, 9210, 9211, 9231, 9233, 9234, 9240, 9242.

STOMACH

Peptic Ulcers

8902 VALUE OF THE ^{14}C-GLYCOCHOLATE TEST: INVESTIGATIONS IN PATIENTS WITH BILLROTH-II-RESECTED STOMACHS. (Ger.) Adlung, J.; Raguse, G.; Grazikowske, H. (I. Medizinische Klinik, Medizinische Hochschule Lubeck, Kronsforder Allee 71/73, D-2400 Lubeck, W. Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 38(1):2-7; 1978.

The ^{14}C-glycocholate exhalation test was performed in 34 healthy subjects, 9 patients with Billroth II gastric resections and various gastrointestinal diseases (fatty degeneration of the liver, chronic pancreatitis, pernicious anemia, dumping syndrome, jaundice, cholelithiasis, cholestasis, malabsorption, and afferent loop syndrome), and 32 subjects with Billroth II gastric resections but without any gastrointestinal diseases. The ^{14}C-glycocholate was administered as a p.o. dose of 10 µCi of labeled compound (specific activity, 10-20 mCi/mmol). All 41 patients had undergone gastric resection for benign ulcer; the glycocholate test was performed 3-4 weeks after gastric resection in 15 cases and 2-40 yr after surgery in 26 cases. The $^{14}CO_2$ exhalation was normal in the healthy controls and in the 32 subjects with Billroth II gastric resections without gastrointestinal diseases, but it was significantly and pathologically increased ($p<0.001$) in 7/9 patients with Billroth II gastric resections and gastrointestinal diseases. According to the hypothesis that the deconjugation of bile salts is possible only by bacterial enzymes, the pathological test result found in the seven patients can be explained only by the presence of bacteria in the upper segment of the intestinal tract or by the loss of bile salts in these patients.

8903 POSTOPERATIVE ANASTOMOTIC ULCER: A REPORT OF 50 CASES. (Fre.) Boiselle, J.; de Montalier, H. (Hopital Bichat, 170, boulevard Ney, 75018 Paris, France). *Ann. Chir.* 31(8):685-692; 1977.

Postoperative ulcers (termed peptic ulcers or anastomotic ulcers) were studied in 50 patients who had previously undergone ulcer surgery. The average delay in appearance of the second ulcer was as long as 30 yr but was only 4 yr for those with previous gastrectomies and 12 yr for those with previous gastroenterostomies. In 34 patients, the anastomotic ulcers could be attributed to technical errors in the initial surgery, usually incomplete vagotomies (11) or insufficient gastrectomies (21). Patients with previous gastroenterostomies underwent a vagotomy and antrectomy or 2/3 gastrectomy as treatment of the second ulcer. Vagotomies were performed on patients with previous gastrectomies and new vagotomies and antrectomies were done for patients who had initially undergone vagotomies

and drainage. Zollinger-Ellison syndrome was found in two patients who had initially been misdiagnosed; they were treated by total gastrectomies. In three patients who developed peptic ulcers after bypass surgery for pancreatic tumors, a modification of the bypass circuit and a new gastroenterostomy were performed. Postoperative mortality was 8/50 but second recurrences occurred in only survivors. It is stressed that successful ulcer treatment must reduce gastric secretion.

8904 THE DEVELOPMENT OF MUCOSAL CHANGES AFTER GASTRIC SURGERY FOR ULCER DISEASE. (Eng.) Janunger, K. G.; Domellof, L.; Eriksson, S. (Univ. Hosp., Umea, Sweden). *Scand. J. Gastroenterol.* 13 (2):217-223; 1978.

To investigate the relationship between morphological changes occurring after gastric surgery and factors such as age, sex, and time of follow-up, and between morphological changes and the development of stump carcinoma, the development of morphological changes in the body mucosa after partial gastrectomy for ulcer disease was studied early (within 3 yr) and late (10-24 yr) postoperatively in 306 patients. Forty-one patients were studied early after Billroth I resection, 14 early after Billroth II resection, 79 late after Billroth I resection, and 172 late after Billroth II resection for gastric or duodenal ulcer. All patients underwent gastroscopy and six forceps biopsies. When agreement between the different biopsies in the same patient was studied, it was not possible to evaluate 19% of the biopsies for technical reasons. On the average, atrophy was found in 4.4 ± 1.5/6 biopsies. The corresponding figures were 4.6 ± 1.5 for acute gastritis, 4.4 ± 1.5 for chronic gastritis, 2.9 ± 1.6 for cystic dilatation, and 1.8 ± 1.2 for intestinal metaplasia. Duodenal ulcer patients and gastric ulcer patients developed mucosal atrophy, cystic dilatation of the gastric glands, and intestinal metaplasia to a similar extent. There was a significantly greater incidence of cystic dilatation of the gastric glands ($p<0.001$) after Billroth II than after Billroth I resection in the patients in whom the biopsy was taken late after surgery. In the early postoperative Billroth I group, an increased incidence in mucosal atrophy, severe chronic gastritis, and intestinal metaplasia were found with increasing age. However, no difference was found in the late postoperative group, and sex did not affect mucosal changes. There were no further relationships between age or type of operation on the development of mucosal changes. Mucosal atrophy, chronic gastritis, and intestinal metaplasia are regarded to be possible features of malignant transformation. If this concept is true, the present findings may explain why stump carcinoma is frequently reported with an incidence that is independent of preoperative ulcer disease or anastomotic procedure.

8905 GASTROESOPHAGEAL SPHINCTER PRESSURE AND SERUM GASTRIN STUDIES FOLLOWING FOOD INTAKE BEFORE AND AFTER VAGOTOMY FOR DUODENAL ULCER. (Eng.) Csendes, A.; Oster, M.; Brandsborg, O.; Moller, J.; Brandsborg, M.; Amdrup, E. (Hospital J. J. Aquirre, Santiago, Chile). *Scand. J. Gastroenterol.* 13(4):437-441; 1978.

The interrelationship between serum gastrin concentration and gastroesophageal sphincter pressure (GESP) was studied in the basal state and after food stimulation in six patients with duodenal ulcer, six patients who had undergone parietal cell vagotomy (PCV), six patients who had undergone selective gastric vagotomy with drainage (SGV + D), and six patients who had undergone SGV and antrectomy (SGV + A). No correlation between GESP and the serum gastrin concentration was observed in the basal state. Immediately after food ingestion, a sustained rise in GESP occurred in the duodenal ulcer patients, which lasted at least up to the end of the experiment. GESP after food ingestion at 60 to 100 min was significantly higher than basal values ($p<0.05$). No increase in GESP occurred in any of the operated patients; in fact, a slight decrease occurred. This decrease was not significantly different from basal values, but was significantly different from the GESP values for duodenal ulcer patients from 60 to 120 min after food ingestion ($p<0.01$). Patients with SGV + D showed basal serum gastrin concentrations that were significantly higher than those in duodenal ulcer patients or patients with SGV + A ($p<0.01$), but not different from those of the PCV patients. After food ingestion, an increase in the serum gastrin concentration occurred in all patients with duodenal ulcer as well as in patients after PCV or SGV + D. Patients with SGV + A showed no increase in gastrin level at any period after food ingestion. The mean gastrin concentration was significantly lower at any period of time compared with the values for all other groups ($p<0.01$). Patients with SGV + D showed significantly greater serum gastrin concentration than did duodenal ulcer patients at 15, 60, and 120 min after food ingestion ($p<0.05$). The increase in gastrin level lasted in all groups for at least 120 min after food intake. The results do not suggest that the serum gastrin concentration and extrinsic vagal innervation in the resting state play a significant role in the maintenance of the tone of the gastroesophageal sphincter. After food ingestion, an interaction may occur between intact vagal innervation and a rise in serum gastrin concentration to obtain an adequate rise in GESP.

8906 PROXIMAL SELECTIVE VAGOTOMY: INTRAOPERATIVE CONTROL OF COMPLETENESS. (Ger.) Jacobs, G.; Baumgartner, H.; Feifel, G.; Martinoli, S. (Chirurgische Klinik A, Universitat Dusseldorf, D-4000 Dusseldorf, W. Germany). *Langenbecks Arch. Chir.* 345:217-222; 1977.

The intraoperative control of the completeness of proximal selective vagotomy (PSV) by the modified vagomotor electrotest according to Burge in the treatment of peptic gastroduodenal ulcer is described. The vagomotor electrotest is based upon the loss of motility of the denervated part of the stomach during electrical stimulation of the vagal

trunks. The denervated part of the stomach is separated from the antrum by a soft clip, and this part of the stomach is filled with air to a pressure of 60-80 mm H_2O and then connected to a pressure recorder. The PSV is complete if the electrical stimulation of the denervated part of the stomach causes a pressure rise. The electrotest was performed in 728 patients; the results were evaluable in 699. Simultaneous pH measurements, performed in 678 cases, were evaluable in 441 cases only. The pH test was positive in 68 cases; the results of the pH test and electrotest coincided in 208 cases. Incomplete PSV was detected only by the pH test in 22 cases, while the pH test was negative in 221 cases in which the electrotest demonstrated incomplete PSV. The findings indicate that the electrotest should always be performed during PSV, regardless of how experienced the surgeon is.

8907 PEPTIC ULCER SURGERY. (Eng.) De Orio, A. J.; Greenlee, H. B. (Veterans Admin. Hosp., Hines, IL 60141). *Surg. Clin. North Am.* 57(6):1159-1172; 1977.

8908 RESULTS OF ONE HUNDRED HIGHLY SELECTIVE VAGOTOMY SIX MONTHS TO FIVE YEARS AFTER OPERATION. (Eng.) Picaud, R.; Baki, N.; Guidicelli, C.; Anfossi, G.; Dalmas, H. (Hopital Nord, Marseille, France). *Chir. Gastroenterol.* 11(2): 173-176; 1977.

8909 ANTRECTOMY-VAGOTOMY VERSUS HIGHLY SELECTIVE VAGOTOMY. (Eng.) De Vries, B. C.; Muller, H.; Cooke, M.; Rogston, J.; Spencer, J.; Dorricott, N.; Alexander-Williams, J. (Univ. Hosp., Rotterdam, Netherlands). *Chir. Gastroenterol.* 11(2):167-171; 1977.

8910 MAGNESIUM AND GASTRIC ACID SECRETION. (Eng.) Puscas, I.; Nadaban, F.; Voicu, L.; Lerintiu, A.; Kovacs, T.; Blaj, St.; *et al.* (Simleu-Silvaniei-Hosp., Simleu-Silvaniei, Rumania). *Acta Hepatogastroenterol. (Stuttg.)* 24(4):288-292; 1977.

8911 PROSPECTIVELY RANDOMISED TRIAL OF PROXIMAL GASTRIC VAGOTOMY EITHER WITH OR WITHOUT PYLOROPLASTY IN TREATMENT OF UNCOMPLICATED DUODENAL ULCER. (Eng.) Wastell, C.; Colin, J.; Wilson, T.; Walker, E.; Gleeson, J.; Zeegen, R. (Westminster Hosp., London SW1P 2AP, England). *Br. Med. J.* 2(6091):851-853; 1977.

8912 SELECTIVE INTUBATION OF THE AFFERENT LOOP. (Eng.) Ho, C. S.; Lipinski, J. K. (Toronto General Hosp., 101 College St., Toronto, Ontario M5G 1L7, Canada). *Am. J. Roentgenol.* 130(3):481-484; 1978.

8913 CIMETIDINE AND SERUM PROLACTIN. (Eng.) Majumdar, S. K.; Thomson, A. D.; Shaw, G. K. (Greenwich District Hosp., London SE10, England). *Br. Med. J.* 1(6110):409; 1978.

8914 EFFECT OF A NEW ANTACID ON BASAL AND MEAL-STIMULATED GASTRIC SECRETION. (Eng.) Sun, D. C. H.; Rider, J. A. (Good Samaritan Hosp., 1033 E. McDowell Rd., Phoenix, AZ 85006). *Am. J. Gastroenterol.* 69(3, Part 1):283-288; 1978.

8915 GIANT DUODENAL ULCER: A DANGEROUS VARIANT OF A COMMON ILLNESS. (Eng.) Klamer, T. W.; Mahr, M. M. (Univ. Louisville Sch. Medicine, Louisville, KY 40201). *Am. J. Surg.* 135(6):760-762; 1978.

8916 SECRETIN SECRETION IN PATIENTS WITH DUODENAL ULCER, CHRONIC PANCREATITIS, AND DIABETES MELLITUS. (Eng.) Ohara, H.; Inabe, S.; Yabu-Uchi, S.; Ohta, H.; Wada, T. (Sapporo Medical Coll., S-1, W-16, Chuoh-ku, Sapporo City, 060 Japan). *Gastroenterol. Jpn.* 13(1):21-27; 1978.

8917 INHIBITION OF GASTRIC SECRETION BY INTRADUODENAL HYPERTONIC GLUCOSE IN PATIENTS WITH DUODENAL ULCER. (Eng.) Hirata, T.; Tani, M.; Yamagishi, T.; Shimazu, H. (Faculty Medicine, Univ. Tokyo, 3-1 Hongo 7, Bunkyo-ku, Tokyo, 113 Japan). *Gastroenterol. Jpn.* 13(1):28-32; 1978.

8918 INTERRELATIONSHIPS BETWEEN THE CHOLINERGIC INFLUENCES, GASTRIC MUCOSA Na^+-K^+-DEPENDENT ATPase, ATP, ADP, IONS OF GASTRIC JUICE AND BASAL SECRETION IN PATIENTS. (Eng.) Mozsik, G.; Kutas, J.; Nagy, L.; Tarnok, F.; Vizi, F. (Univ. Medical Sch., H-7643 Pecs, Hungary). *Acta Physiol. Scand.* (Special Suppl.):199-208; 1978.

8919 DUODENAL ULCER AND WORKING-CLASS MOBILITY IN AN AFRICAN POPULATION IN SOUTH AFRICA. (Eng.) Segal, I.; Dubb, A. A.; Tim, L. O.; Solomon, A.; Sottomayor, M. C. C. G.; Zwane, E. M. (Baragwanath Hosp., Johannesburg, South Africa). *Br. Med. J.* 1(6111):469-472; 1978.

8920 BASAL AND STIMULATED GASTRIN IN DUODENAL ULCER PATIENTS BEFORE AND AT DIFFERENT INTERVALS AFTER HIGHLY SELECTIVE VAGOTOMY (H.S.V.). (Eng.) Lezoche, E.; Marinaccio, F.; Materia, A.; Imperati, L. (III[a] Patologia Chirurgica, Universita di Roma, Rome, Italy). *Chir. Gastroenterol.* 11(3): 283-286; 1977.

8921 LATE CLINICAL RESULTS OF VAGOTOMY, ANTRECTOMY AND GASTRODUODENOSTOMY AT THE LESSER CURVATURE FOR THE TREATMENT OF PEPTIC ULCER. (Eng.) Da Silva, A. L.; Meyer, T. N.; Borges, T. J. A. (Universidade Federal de Minas

Gerais, Sch. Medicine, Belo Horizonte, Brazil). *Chir. Gastroenterol.* 11(3):291-295; 1977.

8922 A STUDY OF THE VARIATIONS IN THE RESPONSE REGARDING DUODENAL ULCER WHEN TREATED WITH PLACEBO BY DIFFERENT INVESTIGATORS. (Eng.) Sarles, H.; Camatte, R.; Sahel, J. (Unite de Recherches de Pathologie Digestive, U-31 INSERM, 46, chemin de la Gaye, F-13009 Marseille, France). *Digestion* 16(4): 289-292; 1977.

8923 CHANGES IN GASTRIC SECRETION FOLLOWING ADMINISTRATION OF PREPARATIONS AFFECTING THE PARASYMPATHETIC AND SYMPATHETIC NERVOUS SYSTEM. (Eng.) Abasov, I. T.; Iof, I. M. (Azerbaijan Gastroenterology Center, Baku, USSR). *Acta Hepatogastroenterol. (Stuttg.)* 25(2):144-149; 1978.

8924 CLINICAL USE OF ENDOSCOPIC CONGORED METHOD IN THE FOLLOW-UP OF PATIENTS WITH SELECTIVE PROXIMAL VAGOTOMY. (Eng.) Suzuki, H. (Jikei Univ. Sch. Medicine, Tokyo, Japan). *Therapiewoche* 28(8):1621-1624; 1978.

8925 CONVENTIONAL MEDICAL MANAGEMENT OF PEPTIC ULCERATION. (Eng.) Crean, G. P. (Southern General Hosp., Glasgow, Scotland). *Scott. Med. J.* 22(4):288-289; 1977.

See also, 8691, 8821, 8855, 8865, 8878, 8882, 8889, 9006, 9210, 9218, 9242.

SMALL INTESTINE

8926 ANGIOGRAPHIC DIAGNOSIS OF INTESTINAL CARCINOID TUMORS. (Ger.) Bardach, G.; Fasching, W.; Raff, M. (Abt. Rontgen, II. Chir. Univ.-Klinik Wien, Spitalgasse 23, A 1097 Vienna, Austria). *Roentgenblaetter* 31(3):155-159; 1978.

Two angiographically documented cases of intestinal carcinoid tumor are described. Carcinoid tumor of the terminal ileum with hepatic metastases was found in the first case and cecal carcinoid tumor without distant metastases in the second. A fibroplastic reaction was found in the mesentery in both cases. The carcinoid tumor in the second patient was strikingly hypervascularized, while most of the primary carcinoid tumors so far described in the literature were hypovascularized. The fibroplastic reaction of the mesentery was indicative of lymph node metastases in both cases. Differentiation between small intestinal carcinoid tumor and Crohn's disease is difficult and often impossible, because mesenteric adhesions seen in Crohn's disease may imitate the typical fibroplastic reaction seen in cases of carcinoid tumor.

8927 ANGIOGRAPHY IN THE DIAGNOSIS OF ILEOCECAL TUBERCULOSIS. (Eng.) Herlinger, H. (St. James' Univ. Hosp., Leeds, England). *Gastrointest. Radiol.* 2(4):371-376; 1978.

To illustrate the use of angiography in the differentiation of ileocecal tuberculosis from Crohn's disease, two case studies are presented. The young women patients presented with pyrexia, abdominal pain, and weight loss. Each had a tender right iliac fossa mass and normal chest x-ray. Barium studies in both patients were consistent with Crohn's disease, showing in case 1, a grossly ulcerated cecum and an irregularly narrowed terminal ileum surrounded by a soft tissue mass and, in case 2, contraction and ulceration of the cecum with the terminal ileum slightly dilated with retained fluid material. Very abrupt demarcation between areas of normality and disease were considered unusual. Angiographic findings (including intense vascularity, small artery irregularity, an intensified venous phase, and in case 1, a positive zoning sign) were typical of Crohn's disease. However, in both cases, an enlarged and hyperemic mesenteric lymph node close to the cecum was clearly shown in the arterial and venous phases of the angiograph. Resection and anastomosis was carried out successfully in both cases and confirmation of the ileocecal tuberculosis was obtained at biopsy of the bowel and regional lymph nodes. The incidence of ileocecal tuberculosis, particularly in underdeveloped countries, the similarities and contrasts between ileocecal tuberculosis and Crohn's disease, the importance of accurate diagnosis, and diagnostic problems are discussed. The angiographic appearance demonstrated in these two patients is a useful diagnostic indicator of tuberculosis etiology. If the findings are confirmed in a larger group, medical antituberculosis therapy might be started without prior biopsy surgery.

8928 EFFECT OF CODEINE PHOSPHATE, LOMOTIL, AND ISOGEL ON ILEOSTOMY FUNCTION. (Eng.) Newton, C. R. (St. Mark's Hosp., London, England). *Gut* 19(5):377-383; 1978.

The effect on ileostomy output of codeine phosphate (C), Lomotil (L), and Isogel (I) was studied in 18 ileostomists living a normal life at home. During a 3-day test period, five subjects took C (60 mg, t.i.d.), seven took L (diphenoxylate, 2.5 mg and atropine, 0.025 mg/tablet, 2 tablets t.i.d.), and

six took I (Ispaghula husk, 15 ml or 6 g, t.i.d.). On one of the test days, a mouth-to-stoma transit study was conducted. Total ileostomy output and output of water were significantly increased by I (+250 g/24 hr, $p<0.01$), significantly decreased by C (-236 g/24 hr, $p<0.05$), and decreased by L (-57 g/24 hr). The mean weight of fecal solids was significantly increased by I (19 g/24 hr, $p<0.01$) but was not significantly changed by C or L. The proportion of fecal solids was unchanged by I or L but was significantly increased by C (2% increase, $p<0.05$). The ileostomy outputs of sodium and potassium increased ($p<0.01$) in response to I, but decreased in response to C and L ($p<0.05$) without change in their concentrations. The output of fecal fat rose significantly in response to C (from 6.1 to 9.6 g/24 hr, $p<0.05$). The total ileostomy output ranged from 383 to 1,335 g/24 hr in 16 of the patients. Two patients on L had total ileostomy outputs greater than 3 kg/24 hr. The effect of L in these patients was similar to its effect in patients with lower total outputs. The excessive ileostomy output was related to extensive ileal resection in these patients. The mouth-to-stoma transit study showed no significant changes due to I or L. In the patients using C, the transit was slower, although not statistically significant, in four of the five subjects. The possible reasons for this negative correlation between C and transit time are discussed along with the reasons why L appears to be less effective than C in the present study. The authors conclude that C and L are effective in reducing the ileostomy loss of water and electrolytes, while I increases their loss and can aggravate depletion in patients with excessive fluid effluents. The increase in fecal fat associated with C use suggests that it should be stopped before collecting samples for fecal fat determinations.

8929 RADIOLOGIC EVALUATION OF THE CONTINENT (KOCK) ILEOSTOMY. (Eng.) Montagne, J. P.; Kressel, H. Y.; Moss, A. A.; Schrock, T. R. (Univ. California Sch. Medicine, San Francisco, CA). *Radiology* 127(2):325-329; 1978.

The surgical procedure for constructing an ileostomy with an ileal reservoir, a continent (Kock) ileostomy, and the radiographic features of normal and malfunctioning continent ileostomies are described. In the surgical procedure, approximately 30 cm of ileal loop is sutured to itself, the bowel is cut, and the ileal loop is opened. The reservoir is manufactured by folding and suturing the distal part of the loop to the proximate end. A nipple valve is created by intussusception of 4-5 cm of distal ileum into the lumen of the reservoir. Radiographically, the normal ileal reservoir is usually seen as a slightly lobulated gas-filled structure in the middle or right side of the pelvis. Unemptied, the reservoir may simulate a pelvic abscess on plain film. Using a contrast agent administered p.o. or retrograde by means of an ileostomy enema, the oval shape of the ileal reservoir and the fine, feathery, nonedematous appearance of the ileal mucosa in the reservoir and the proximal ileal segment are seen. The terminal invaginated ileal segment resembles an inverted nipple protruding into the pouch. This normal finding should not be mistaken for a mass or intussusception. Absence of this filling defect indicates prolapse of the nipple valve out of the nipple pouch, and surgical revision is required. The site of a small bowel obstruction can be determined by means of either small bowel follow-through examination or an ileostomy enema administered retrograde with reflux into the proximal ileum. When abscess formation is suspected, an abdominal radiograph should be made after the reservoir has been drained; the abscess will remain visible in the emptied reservoir. If anastomotic breakdown is suspected, a radiographic examination using water-soluble, iodinated contrast medium administered retrograde through the ileostomy will demonstrate extravasation of contrast medium outside the reservoir. Nonspecific ileitis and recurrent granulomatous disease of the ileal reservoir or proximal ileum show radiographically irregular, thickened, blunted mucosal folds in the pouch and ileum. Endoscopy and mucosal biopsy may be needed to detect a recurrence of Crohn's disease.

8930 DUODENAL MUCOSAL DAMAGE ASSOCIATED WITH CHRONIC USE OF ANTI-INFLAMMATORY DRUGS. (Eng.) Tanaka, M.; Uchiyama, M.; Ikeda, S. (Higashi-Kunisaki Koiki Hosp., 1456 Shimoharu, Aki-cho, Higashi-Kunisaki-gun, Oita 873-02, Japan). *Endoscopy* 9(3):136-139; 1977.

Three cases of multiple duodenal erosions or ulcers associated with the long-term use of anti-inflammatory agents are presented. The first patient was a 58-yr-old man who had ingested various analgesics (ibuprofen, diclofenac sodium with aminopyrin, phenacetin, and caffeine; buffered aspirin with aminopyrin, phenacetin, and caffeine; and buffered aspirin with aminopyrin, phenacetin, and caffeine) for various periods over 15 months for spastic muscle aches associated with quadriplegia due to traumatic cervical cord injury. The patient developed massive hematemesis and melena, which ceased after 10 days of treatment; this consisted of withdrawal of p.o. analgesics, bland diet, and antacids. The bleeding was from multiple large ulcers involving the bulbar and postbulbar portion of the duodenum. The antrum also contained a few erosions. The second patient was a 67-yr-old man who had taken 2.0 g of buffered aspirin p.o. in addition to a rectal dose of 50 mg of indomethacin daily for 10 months to reduce arthralgia. He presented with epigastralgia accompanied by hematemesis. Endoscopy revealed diffuse superficial gastritis with multiple hemorrhagic erosions, which were most numerous in the antrum. Shallow, actively bleeding erosions were seen in the duodenum. The third patient, a 47-yr-old woman who had taken ibuprofen (800 mg/day, p.o.) and indomethacin (50 mg/day, rectally) for 10 weeks, presented with hematemesis. Endoscopy showed extensive erosive gastritis with shallow ulceration. Marked congestion, friability and multiple shallow ulcers were seen in the duodenal bulb. This report emphasizes the possibility of duodenal mucosal injury related to anti-inflammatory agents.

8931 BYPASS SURGERY FOR OBESITY. (Eng.) Sorrell, V. F. (Middlemore Hosp., Otahuhu, Auckland, New Zealand). *Aust. N.Z. J. Surg.* 47(5): 656-659; 1977.

8932 IMPROVEMENT IN QUALITY OF LIFE FOLLOWING JEJUNOILEAL BYPASS SURGERY FOR OBESITY. (Eng.) Dano, P.; Hahn-Pedersen, J. (Rigshospitalet, Copenhagen, Denmark). *Scand. J. Gastroenterol.* 12(7):769-774; 1977.

8933 CHRONIC INTESTINAL PSEUDOOBSTRUCTION. (Eng.) Faulk, D. L.; Anuras, S.; Christensen, J. (Univ. Iowa Hosp., Iowa City, IA 52242). *Gastroenterology* 74(5, Part 1):922-931; 1978.

8934 ILEAL DUPLICATION CYST: THE IMPORTANCE OF SODIUM PERTECHNETATE Tc 99m SCANNING. (Eng.) Rose, J. S.; Gribetz, D.; Krasna, I. H. (Mount Sinai Hosp., 100th St. and Fifth Ave., New York, NY 10029). *Pediatr. Radiol.* 6(4):244-246; 1978.

8935 GASTROINTESTINAL HEMORRHAGE FROM ILEAL LEIOMYOMA: UTILITY OF THE COMPLETE REFLUX SMALL BOWEL EXAMINATION. (Eng.) Miller, R. E.; Lehman, G. (Indiana Univ. Medical Center, 1100 W. Michigan St., Indianapolis, IN 46202). *Gastrointest. Radiol.* 2(4):367-369; 1978.

8936 RELATIONSHIP BETWEEN DIETARY CALCIUM AND HYPEROXALURIA AFTER INTESTINAL SHUNT OPERATION FOR OBESITY. (Eng.) Hessov, I.; Holm, C. N.; Hansen, L. P. (Kommunehospitalet, 8000 Arhus C, Denmark). *Digestion* 17(3):268-271; 1978.

8937 FALSE POSITIVE SCAN FOR MECKEL DIVERTICULUM. (Eng.) Tauscher, J. W.; Bryant, D. R.; Gruenther, R. C. (Michigan State Univ. Coll. Human Medicine, East Lansing, MI). *J. Pediatr.* 92(6):1022-1023; 1978.

8938 TECHNIQUE OF SCANNING FOR MECKEL'S DIVERTICULUM. (Eng.) Singh, P. R.; Russell, C. D.; Dubovsky, E. V.; Bridger, W. M. (Univ. Alabama Hosp., Birmingham, AL). *Clin. Nucl. Med.* 3(5):188-192; 1978.

8939 RECURRENT INTUSSUSCEPTION: ANALYSIS OF A SERIES TREATED WITH HYDROSTATIC REDUCTION. (Eng.) Eklof, O.; Reiter, S. (Karolinska Sjukhuset, S-104 01 Stockholm, Sweden). *Acta Radiol. [Diagn.] (Stock.)* 19(1B):250-258; 1978.

8940 JEJUNO-ILEAL BYPASS FOR MORBID EXOGENOUS OBESITY. (Eng.) Balfour, J. F. (No affiliation given). *Hawaii Med. J.* 37(1):9-15; 1978.

8941 INTRAOPERATIVE LOCALIZATION OF INTESTINAL ARTERIOVENOUS MALFORMATION. (Eng.) Evans, W. E.; O'Dorisio, T. M.; Molnar, W.; Martin, E. W., Jr.; Wooley, C. F.; Cooperman, M. (Ohio State Univ. Hosp., 410 W. Tenth Ave., Columbus, OH 43210). *Arch. Surg.* 113(4):410-412; 1978.

8942 HYPERPLASIA OF BRUNNER'S GLANDS, POLYPOID DUODENAL PATTERN AND DUODENITIS. (Eng.) Maratka, Z.; Kocianova, J.; Kudrmann, J.; Pirk, F. (Bulovka Hosp., 18081 Praha 8, Czechoslovakia). *Rend. Gastroenterol.* 9(3):202-209; 1977.

8943 MULTIPLE SMALL BOWEL STRICTURES IN A CHILD AND ACCIDENTAL POTASSIUM CHLORIDE INGESTION. (Eng.) Tresadern, J.; Rickwood, A. M. K.; Spitz, L. (Children's Hosp., Sheffield S10 2TH, England). *Br. Med. J.* 2(6095):1124-1125; 1978.

8944 BOUVERET'S SYNDROME. (Eng.) Sullivan, K. P.; Clemett, A. R.; Ferrara, T. P. (St. Vincent's Hosp., New York, NY). *Am. J. Gastroenterol.* 68(4):399-404; 1977.

8945 UNDERSTANDING THE BOWEL OBSTRUCTION PROBLEM. (Eng.) Wangensteen, O. H. (Univ. Minnesota Medical Sch. Complex, Box 610, Minneapolis, MN 55455). *Am. J. Surg.* 135(2):131-147; 1978.

8946 ENTEROCUTANEOUS FISTULAS OF THE SMALL BOWEL IN CHILDREN (25 CASES). (Eng.) Nihoul-Fekete, C.; Ricour, C.; Duhamel, J. F.; Lecoultre, C.; Pellerin, D. (Hopital des Enfants Malades, Paris, France). *J. Pediatr. Surg.* 13(1): 1-4; 1978.

See also, 8625, 8775, 8781, 8794, 8796, 8805, 8807, 8810, 8811, 8812, 8817, 8819, 8821, 8828, 8842, 8879, 8893, 8894, 8907, 8912, 8949, 8955, 8966, 8992, 8998, 9006, 9047, 9175, 9178, 9192, 9195, 9211, 9227, 9230, 9231, 9233, 9235, 9240, 9247, 9262.

8947 THE ROLE OF VARIOUS CEREALS IN COELIAC DISEASE. (Eng.) Anand, B. S.; Piris, J.; Truelove, S. C. (Radcliffe Infirmary, Oxford, England). *Q. J. Med.* 47(185):101-110; 1978.

The effects of four different cereals (60 g/day, p.o., for 1 month) in celiac disease were assessed in celiac patients in remission on a gluten-free diet. Jejunal biopsy in all five patients who received barley revealed definite evidence of histological damage, yet none of the patients experienced symptoms. The mean surface to volume ratio before barley consumption was 30.48, and this decreased considerably after barley consumption in all but one patient who had a low ratio to start with. There was a fall in disaccharidase levels in all five patients ($p<0.05$ for mean levels of sucrase, lactase, and maltase). Two patients received rye. In the first patient, the jejunal biopsy taken before rye consumption showed partial villous atrophy, whereas the other patient had a near-normal jejunal mucosa at the start of the study. After rye consumption, the biopsy showed an almost complete loss of villi, crypt hypertrophy, and an increase in chronic inflammatory cells in the lamina propria. The surface to volume ratio decreased from 22.19 to 12.79. The second patient developed diarrhea, nausea, abdominal discomfort, and irritability after consuming rye for 3 weeks. The villi were short and broad, there was crypt hyperplasia with an increase in the number of mitotic cells, the inflammatory infiltrate had increased, and the superficial epithelial cells were flat with loss of polarity of the nuclei. The surface to volume ratio decreased from 48.64 to 29.20. All three disaccharidases decreased in biopsy specimens from both patients. Neither of two patients who consumed maize and none of three who consumed rice developed symptoms or histological abnormalities in the jejunum. The results indicate that barley and rye products should be eliminated from the diets of patients with celiac disease.

8948 VARIATIONS IN THE BLOOD AMINO ACID LEVELS IN CHILDREN WITH CELIAC DISEASE FOLLOWING SPECIAL DIET THERAPY. (Ita.) Balli, F. (Istituto di Clinica Pediatrica dell'Universita di Modena, Modena, Italy). *Minerva Pediatr.* 29(37):2255-2257; 1977.

Data concerning 13 children (aged 8 months-6 yr) with celiac disease are presented. The symptoms included biological and clinical signs of malabsorption when the diet contained gluten, typical histological abnormalities of the duodenal-jejunal mucosa, and clinical and biological remission when gluten was withdrawn from the diet. All of the children had grade III atrophy of the jejunal mucosa. The phase of the disease was determined by the gas chromatography profile of the levels of free amino acids in their sera. The patients' diets were changed: gluten and lactose were excluded, and an enzymatic hydrolysate of casein was added. Children <1 yr old received 5-6 protein/kg, 12-15 g carbohydrates (glucose, fructose from honey, starch from rice, corn, tapioca, and sucrose from fruit)/kg, and 2.5-3 g of fat/kg daily. Before the diet therapy, the levels of the essential amino acids (particularly valine, isoleucine, lysine, and phenylalanine) were low, while the nonessential amino acids (particularly glycine) were elevated. After only 1 month of diet therapy, while clinical results were not yet evident, there was an increase in the blood levels of several of the essential amino acids (approximate average values were: valine increased from 2 to 5 mg%, isoleucine from 1 to 2 mg%, phenylalanine from 3 to 4 mg%, and lysine from 2 to 4 mg%), while leucine decreased from 3 to 2.5 mg%; there was a decrease in the glycine level (from 4 to 2 mg%). The glycine/valine ratio changed from 2.06 before treatment to 0.47 (normal value is <1) after 1 month of diet therapy. Early clinical improvements followed (changes in mood and in psychomotor and neurological functions). The results demonstrate the correlation between normalization of protein metabolism and early improvement in psychomotor and neurological functions.

8949 FUCOSIDOSIS AND I-CELL DISEASE: A FINE-STRUCTURAL AND SILVER-STAINING STUDY OF ABNORMAL INCLUSION BODIES IN SMALL-INTESTINAL CELLS. (Eng.) Ginsel, L. A.; Cambier, P. H.; Daems, W. T. (Lab. Electron Microscopy, Univ. Leiden, Rijnsburgerweg 10, Leiden, Netherlands). *Virchows Arch. [Cell Pathol.]* 27(2):99-117; 1978.

Small intestinal cells of a 14.5-yr-old patient with fucosidosis, a 9-month-old patient with I-cell disease, and three control children (ages 2.5, 9, and 11 yr) with suspected familial celiac disease were studied, with special attention given to the fine structure and silver-staining patterns of abnormal, lysosome-like inclusion bodies. Abnormal inclusion bodies were found in the small intestinal cells of both patients. The silver-proteinate affinity of the stored material in the greater part of these abnormal inclusion bodies demonstrated the presence of mucopolysaccharides and/or glycoproteins. The lamellar structures regularly observed in these vacuoles indicated the storage of lipid-like material. Significantly enlarged lysosome-like bodies in the absorptive cells of these two patients showed the same silver-stain affinity as the Golgi apparatus, the apical vesicles and tubules, and the cell coat on the microvilli of the absorptive cells. Fusions between the apical vesicles and tubules and the lysosome-like bodies, all of which had identical silver-proteinate affinity, were also observed. These findings suggest a regulatory function of the lysosome-like bodies in the transport or secretion of cell coat material that might be of the crinophagic type.

The presence of many abnormal vacuoles in the macrophages of both patients and in the endothelial cells of the patient with fucosidosis indicated that these cells too are involved in lysosomal storage. Numerous abnormal inclusion bodies in the fibroblasts in the small intestine of the patient with fucosidosis were also observed. Abnormal inclusions were also found in the fibroblasts in the small intestine of the patient with I-cell disease. The patient with I-cell disease also showed normal inclusion bodies in peripheral blood lymphocytes and cultured skin fibroblasts. The above findings are compatible with the concept of inborn lysosomal storage disease, which explains the accumulation of material in lysosomal storage vacuoles as a consequence of inadequate degradation due to a deficiency of lysosomal hydrolytic enzymes.

8950 COWS' MILK PROTEIN-SENSITIVE ENTEROPATHY: COMBINED CLINICAL AND HISTOLOGICAL CRITERIA FOR DIAGNOSIS. (Eng.) Iyngkaran, N.; Robinson, M. J.; Prathap, K.; Sumithran, E.; Yadav, M. (Dept. Pediatrics, Univ. Malaya, Kuala Lumpur, Malaysia). *Arch. Dis. Child.* 53(1):20-26; 1978.

Diagnostic criteria for cows' milk protein-sensitive enteropathy were assessed in 15 infants with diarrhea, all of whom were suspected of having this type of enteropathy. Cows' milk was eliminated from the diet for 6-8 weeks. A jejunal biopsy was then carried out, and a repeat biopsy was performed 24 hr after challenge with cows' milk formula. The biopsy appearance was classified on the basis of four parameters: villous morphology under the dissecting microscope, villous/crypt height ratio, epithelial cells, and cellular infiltration. In four infants, no significant alteration was noted in the jejunal mucosa 24 hr after the reintroduction of cows' milk; these infants continue to thrive on cows' milk formula. The biopsy in the remaining 11 patients was considered to be moderately abnormal in 8 and severely abnormal in 3, although the prechallenge biopsy was normal in 4 and mildly abnormal in the other 8. Seven of the 11 infants developed symptoms at 0.5 hr to 2 weeks after challenge, but the other 4 have not yet developed symptoms and are making satisfactory clinical progress on a cows' milk formula. The following set of criteria for cows' milk protein-sensitive enteropathy are proposed: (a) clinical disease (diarrhea with or without vomiting) while receiving cows' milk protein, (b) clinical improvement on a diet free of cows' milk protein, (c) normal or mildly abnormal jejunal histology 6-8 weeks after symptoms subside, and (d) histological relapse, with or without clinical relapse, after reintroduction of cows' milk protein.

8951 FIRST OBSERVATION OF COLLAGEN SPRUE IN AN INFANT. (Fre.) Mougenot, J. F.; Martin, E.; Lemoine, P.; Boccon-Gibod, L.; Fontaine, J. L.; Navarro, J.; *et al.* (Hopital Trousseau, 28, rue du docteur A. Netter, F 75571 Paris Cedex 12, France). *Nouv. Presse Med.* 6(30):2689-2690; 1977.

An unusual case of collagen sprue in a 17-month-old infant is reported. The symptoms of sprue followed a bout of infectious diarrhea that affected the whole family, but only this child developed complications. The complications appeared to consist of protein exudative enteropathy, electrolyte depletion with secondary hyperaldosteronism, and intestinal dyskinesia causing repeated partial occlusion. These symptoms are similar to those observed in collagen sprue in adults with the difference that the intestinal lesions (ulcerous ileitis, collagen infiltration of the intestinal mucosa) were more distal in the infant. A gluten-free diet and a diet without allergens failed to improve the condition. An ileal bypass was performed, but the intestinal fluid exudate persisted and p.o. corticotherapy was initiated followed by i.v. steroids. Only slight improvement occurred and the child died at the age of 3.5 yr after a succession of staphylococcal infections resistant to antibiotics. It was thought that the disease may have been related to an immunopathological reaction to microbial antigens of the intestinal flora. A lymphoblast transformation test with coli bacillus antigens was positive, but other tests for immune deficiencies or allergies were negative.

8952 QUANTITATIVE ANALYSIS OF FECAL FAT DURING LIPASE SUBSTITUTION. (Ger.) Sailer, D.; Arnholdt, W.; Berg, G. (Medizinische Universitatsklinik, Postfach 3560, 8520 Erlangen, W. Germany). *Therapiewoche* 27(52):9353-9357; 1977.

8953 A NEW RADIOIMMUNOASSAY FOR DETERMINING NONPRECIPITATING ANTIBODIES TO GLIADIN IN CELIAC DISEASE AND DERMATITIS HERPETIFORMIS. [Abstract]. (Ger.) Menzel, E. J.; Granditsch, G.; Pehamberger, H.; Winterleitner, H.; Molinari, E. (Institut fur Immunologie, Universitat Wien, Vienna, Austria). *Z. Immunitaetsforsch.* 153(4):333; 1977.

8954 THE SYMPTOMS OF CELIAC SPRUE (CELIACA) IN ADULTS. (Dut.) Cluysenaer, O. J. J.; van Tongeren, J. H. M. (St. Radboud Ziekenhuis, Nijmegen, Netherlands). *Ned. Tijdschr. Geneeskd.* 121(3):87-95; 1977.

8955 LACTASE ACTIVITY AND THE MILK INTOLERANCE SYNDROME. (Rus.) Valenkevich, L. N. (Leningrad Inst. Pediatric Medicine, Leningrad, USSR). *Klin. Med. (Mosk.)* 55(2):66-70; 1977.

8956 ENZYME SUBSTITUTION THERAPY OF SECONDARY LACTOSE MALABSORPTION. (Hun.) Frank, K.; Barna, M. (Gyermekgyogyaszati Klinika, Orvostovabbkepzo Intezet, Budapest, Hungary). *Orv. Hetil.* 118(35):2093-2095; 1977.

8957 NONTROPICAL SPRUE. (Ger.) Heilmann, E.; Simpson, B. (Medizinischen Poliklinik der Universitat, Westring 3, 4400 Munster, W. Germany). *Therapiewoche* 27(42):7523-7526; 1977.

8958 CLINICAL PICTURE OF WHIPPLE'S DISEASE. (Ger.) Leinweber, W. (Klinikum, Johann Wolfgang Goethe-Universitat, Theodor-Stern-Kai 7, 6000 Frankfurt a. M.-70, W. Germany). *Therapiewoche* 27(38):6661-6665, *passim;* 1977.

8959 REPORT OF A CASE OF WHIPPLE'S DISEASE WITH HYPEREOSINOPHILIA. (Fre.) Herreman, G.; Gentilini, M.; Danis, M.; Brucker, G.; Marche, Cl. (Hopital de la Pitie, F 75634 Paris Cedex 13, France). *Ann. Med. Interne (Paris)* 128(12):973-978; 1977.

See also, 8625, 8796, 8810, 8811, 8933, 9071, 9165, 9216, 9223, 9232, 9233, 9239, 9241, 9250, 9254.

LARGE INTESTINE

8960 PSEUDOMEMBRANOUS COLITIS: 10 CASES. (Fre.) Tubiana, J. M.; Nordlinger, D.; Chermet, J.; Douvin, D.; Lecomte, A. (Hopital Saint-Antoine, 184, rue du Faubourg Saint-Antoine, 75571 Paris Cedex 12, France). *J. Radiol. Electrol. Med. Nucl.* 58(12):811-818; 1977.

Ten case histories are presented of patients who developed pseudomembranous colitis after treatment with antibiotics. Lincomycin (1.5-2 g/day, p.o. or 1-2.4 g/day, i.v. or i.m.) and ampicillin (2 g/day, p.o. or 3-12 g/day, i.v.) were involved in five cases each (once in association with each other), staphlomycin (2 g/day, p.o.) in one case, and clindamycin (1.2 g/day, p.o.) and thiophenacol (1.5 g/day, i.m.) each once in association with lincomycin or ampicillin, resp. Duration of treatment ranged from 4-40 days but was usually 5-10 days. The initial clinical sign was diarrhea in every case. Rectoscopy showed a false membrane composed of fibrin, mucous, leukocytes, necrotic cells, and epithelial debris. The colon glands were hypertrophied and hypersecretory. The characteristic appearance of the colon on barium x-ray revealed diffuse "thumbprint" lacunae, 2-4 mm in diameter, which projected slightly into the lumen of the intestine when profile x-rays were taken. The lacunae were scattered throughout the left colon with intervals of healthy mucosa between them. The x-ray signs regressed, often within a month, after the antibiotics were discontinued.

8961 CEPHALOSPORIN-ASSOCIATED PSEUDOMEMBRANOUS COLITIS. (Eng.) Hutcheon, D. F.; Milligan, F. D.; Yardley, J. H.; Hendrix, T. R. (Johns Hopkins Hosp., 601 North Broadway, Baltimore, MD 21205). *Am. J. Dig. Dis.* 23(4):321-326; 1978.

The association of pseudomembranous colitis (PC) with the use of the antibiotic, cephalosporin, was investigated in three cases. In cases 1 and 2, the patients had received pre- and postoperative therapy with cephalothin or cephalexin; in case 3, the patient had been treated with a 5-day course of cephalexin (250 mg, t.i.d.) for an infected hornet bite. In case 1, symptoms of diarrhea and hematochezia with fever were relieved by discontinuation of cephalothin and initiation of i.v. ampicillin and chloramphenicol therapy. In case 2, diarrhea with small amounts of blood developed within a month of hospital discharge. Cholestyramine treatment (2-12 g/day) was required over a period of 9 weeks. In case 3, fever and diarrhea developing within a week of cephalexin treatment responded to cholestyramine treatment (6-12 g daily for 7 days). In case 1, pseudomembranes were not seen sigmoidoscopically, but acute inflammation in the mucosa and inflammatory exudate on the surface were observed histologically. In cases 2 and 3, sigmoidoscopy revealed pseudomembranes, and inflammation was seen histologically. In case 2, the histologically demonstrable inflammation continued after pseudomembranes disappeared sigmoidoscopically. In all cases, stool cultures were negative for pathogens and the diagnosis of PC was confirmed by rectal biopsy. The incidence of cephalosporin-related colitis was shown to be less common than clindamycin colitis but the authors conclude that cephalosporins should be added to the list of antibiotics (chloramphenicol, tetracycline, lincomycin, ampicillin, and clindamycin) associated with PC. They recommend that a biopsy be performed when antibiotic colitis is suspected even when gross inspection reveals only hyperemia and edema. Demonstration of acute inflammation could alert the clinician to the possible development of PC if antibiotic therapy were continued.

8962 PSEUDONEOPLASTIC SIGMOID DIVERTICULITIS: THE VALUE OF I.V. UROGRAPHY IN DIAGNOSIS. (Fre.) Cugnenc, P. H.; Rouanet, J. P.; Loygue, L.; Nouel, O.; Parc, R. (Hopital Saint-Antoine, 184, rue du Faubourg Saint-Antoine, 75571 Paris Cedex 12, France). *J. Radiol. Electrol. Med. Nucl.* 58(12): 801-806; 1977.

In a group of 160 patients treated for sigmoid diverticulitis in the period 1970-1975, the initial diagnosis on the basis of clinical, radiological, and surgical criteria was pseudotumoral sigmoiditis in 63. Further study, particularly x-ray studies, confirmed the diagnosis in 45/63, but the possibility of the presence of cancer was not eliminated preoperatively in the remaining 18. Clinical, radiological, endoscopic, and surgical exploration of these 18 patients is discussed, and a typical case of so-called pseudoneoplastic sigmoiditis is presented. The average age of patients with sigmoiditis is 58 yr, which is close to average age of a series of 258 patients with cancer of the colon, 62 yr. Functional signs do not differ in the two groups, and endoscopy fails to differentiate between cancer and diverticulitis in about 50% of patients. X-ray studies show extensive inflammatory lesions causing changes and thickening in the intestinal muscle wall. Chronic lesions can invade the mesosigmoid, the retrocolic tissue, and neighboring organs, particularly the bladder. Signs of urinary retention, either clinical or observed by i.v. urography, occur in about 20% of patients with sigmoiditis and may be the only symptom in some cases. I.v. urography was performed in 6 of the 18 patients in this series. Typical bladder signs included pseudotumoral changes in the bladder and evidence of external compression, particularly in the area of the left part of the vesical dome, and of retention.

8963 VASCULAR ECTASIAS OF THE RIGHT COLON. (Eng.) Sprayregen, S.; Boley, S. J. (Montefiore Hosp. and Medical Center, Bronx, NY 10467). *JAMA* 239(10):962-964; 1978.

The use of angiography in the diagnosis of vascular ectasias of the right colon is described, and the results in 25 patients are presented. In a patient with lower intestinal tract bleeding, a nasogastric tube should be passed to exclude gastric or duodenal bleeding. If the findings in this investigation and in coagulation studies, sigmoidoscopy, and colonoscopy are normal, emergency angiography of the superior mesenteric artery, inferior mesenteric artery, and celiac axis should be carried out, in that order. The technique involves inserting a rectal tube before arterial catheterization. The superior mesenteric artery is catheterized, and the colon is then distended with air. For this artery, 50 ml of a combination of 10% diatrizoate sodium and 66% diatrizoate meglumine is injected at 8 to 10 ml/sec, and filming is at the rate of one film per second for 12 sec, followed by one film every other second for 16 sec. The following angiographic criteria are reliable and important for the diagnosis of vascular colonic ectasias: (a) a densely opacified, dilated, tortuous, slowly emptying intramural vein that remains visualized after the other mesenteric veins have emptied; (b) a vascular tuft, which is best identified in the arterial phase; (c) an early-filling vein, which can be visualized within 4-5 sec; and (d) intraluminal extravasation of contrast medium, refering to a localized, amorphous collection that first appears during the arterial phase of the arteriogram and persists throughout the study. A densely opacified, dilated, tortuous, slowly emptying intramural vein was noted in 92% of the 25 patients with ectasias, and a vascular tuft was present in 74%. An early-filling vein, indicating the presence of an advanced lesion with arteriovenous shunting, was definitely present in 61% and suggested in another 16%. On the basis of previous studies, the authors conclude that vascular ectasias of the right colon are acquired, degenerative lesions that are common in the aging population.

8964 MULTIPLE CAVERNOUS HEMANGIOMAS OF THE COLON. (Ger.) Wagner, H.; Neugebauer, R.; Bultmann, B. (Rontgendiagnostische Abteilung des Departments Radiologie, Steinhovelstrasse 9, 7900, Ulm/Donau, W. Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 128(2):131-134; 1978.

Cavernous hemangioma of the ascending, transverse, and descending colon; the sigmoid; and the rectum was diagnosed in a 26-yr-old man by rectoscopy, double-contrast rectocoloscopy, and arteriography. The patient was hospitalized for recurrent rectal bleeding and abdominal distention. Arteriography demonstrated vascular tumors in the area of the jejunal artery, the left colonic artery, and the superior rectal artery. This rare condition has been described only 53 times in the literature. Subtotal colectomy with cecorectostomy was successful. The excessively frequent defecation observed after surgery was checked by cholestyramine. The diagnostic importance of the double-contrast examination of the colon and of selective angiography in the differential diagnosis of this rare condition is stressed.

8965 HISTOCHEMICAL CRITERIA FOR THE DIAGNOSIS OF HIRSCHSPRUNG'S DISEASE IN RECTAL SUCTION BIOPSIES BY ACETYLCHOLINESTERASE ACTIVITY. (Eng.) Chow, C. W.; Chan, W. C.; Yue, P. C. K. (Royal Children's Hosp., Flemington Rd., Parkville, Victoria 3052, Australia). *J. Pediatr. Surg.* 12(5): 675-680; 1977.

Rectal suction biopsies taken from 96 patients who presented with chronic constipation or intestinal obstruction were examined to determine the histochemical criteria for diagnosis of Hirschsprung's disease (HD) by acetylcholinesterase (ACh) activity. In 37 of the 96 cases, the diagnosis or exclusion of HD was made by searching for ganglion cells in serial paraffin sections, and histochemical patterns were examined retrospectively to determine the features of a positive and a negative reaction. In the 27/37 cases in which HD was excluded histologically, ACh activity showed staining along the luminal border and around lymphoid tissue and diffuse staining of the muscularis mucosae and of bundles of cholinergic fibers in the deeper portions of the submucosa; occasionally, some fine discrete cholinergic fibers were also seen in the muscularis mucosae and lamina propria. In the 10/37 cases di-

agnosed as HD, there were two types of positive reaction: type A showed coarse discrete cholinergic fibers in the muscularis mucosae and the immediately adjacent submucosa and sometimes in the lamina propria, whereas in type B, the lamina propria was devoid of or contained only a few discrete fibers. The histochemical criterion for a positive reaction was therefore chosen as coarse discrete fibers in the muscularis mucosae and adjacent submucosa. In 59 of the 96 remaining cases, the histochemical reaction was determined first and checked for reliability by histological features. In the 42/59 cases showing a negative reaction, ganglion cells could be seen without difficulty; in the 17/59 cases with a positive reaction, ganglion cells were absent in all but 2. In one of the latter cases, four degenerate ganglion cells were seen in the biopsy taken at 5 days of age; however, no ganglion cells were seen in a repeat biopsy at 7 months. In the second case, a few clusters of well formed ganglion cells were seen in the 5-cm specimen; the absence of ganglion cells in the 3-cm and 4-cm specimens was, however, consistent with the diagnosis of ultrashort segment HD. It is possible that the former case represents an etiology for HD that involves destruction of ganglion cells by viral infection in intra-uterine and early postnatal life.

8966 A SECRETORY EPITHELIUM OF THE SMALL INTESTINE WITH INCREASED SENSITIVITY TO BILE ACIDS IN IRRITABLE BOWEL SYNDROME ASSOCIATED WITH DIARRHOEA. (Eng.) Oddsson, E.; Rask-Madsen, J.; Krag, E. (Herlev Univ. Hosp., DK-2730 Herlev, Denmark). *Scand. J. Gastroenterol.* 13(4):409-416; 1978.

To determine the cause of malabsorption of water and electrolytes in patients with irritable bowel syndrome with associated diarrhea (IBSD), perfusion studies of the ileum and jejunum were performed in 6 patients with IBSD and compared with identical studies in 10 normal subjects. Simultaneous measurements were made of the net movements of water and electrolytes; the lumen to plasma and plasma to lumen fluxes of Na, K, and Cl; and the transmural electrical potential difference (PD) in response to perfusion with glycochenodeoxycholic acid (GCDC) at concentrations of 0.25, 0.5, 1.0, 1.5, and 2.5 mM. In normal subjects, solutions containing 1 mM GCDC stimulated ileal absorption ($p<0.05$), while further increases of bile acid concentration reduced fluid absorption and evoked net secretion. Patients with IBSD showed fluid secretion even when no bile acid was present. Their mean water movement was significantly ($p<0.05$) different from that of normal volunteers. Increased absorption occurred at GCDC concentration of 0.25 mM; concentrations above this level increased secretion. Expressed graphically, the patient curve is seen as a parallel transposition of the normal curve to the secretory side and to lower concentrations of bile acids, suggesting an increased sensitivity of the secretory epithelium to the stimulus of GCDC in the patients. Increased plasma to lumen fluxes appeared to be responsible for the spontaneous secretion of Na and Cl in IBSD. GCDC reduced the lumen to plasma fluxes of Na and Cl equally in both groups. Therefore, the ileal epithelium in IBSD patients responds normally to GCDC, but may be prestimulated by some other biological mechanism involved in intestinal secretion. The jejunal patterns were similar to the ileal. No changes in the transmural PD were demonstrated. The authors foresee therapeutic implications in the conclusions that the small intestinal epithelium in IBSD, besides being prestimulated by some as yet unidentified agent, shows an increased sensitivity to the stimulus of GCDC.

8967 PRIMARY LINITIS PLASTICA OF THE COLON: REPORT OF TWO CASES AND REVIEW OF THE LITERATURE. (Eng.) Amorn, Y.; Knight, W. A. (St. Mary's Health Center, 6420 Clayton Rd., St. Louis, MO 63117). *Cancer* 41(6):2420-2425; 1978.

Two cases of primary linitus plastica of the colon (scirrhous carcinoma of the signet ring cell type) are reported along with a review of 20 cases in the literature. A 61-yr-old man had a two-month history of bilateral cramping abdominal pain and diarrhea, a 20-pound weight loss, and five to six watery stools/day containing occult blood. Colonoscopy demonstrated a stricture of the sigmoid colon at 40 cm and a 1-cm polyp distal to the stricture. An air contrast barium enema showed a 5-cm constriction in the mid-sigmoid area with overhanging edges at both ends. Sigmoid resection with end-to-end anastomosis of the colon was performed. A 20-yr-old man had a 3-month history of diarrhea, anorexia, a 20-pound weight loss, and loose watery stools numbering 6-12/day and containing gross and occult blood. Rectal examination showed an indurated area 2-3 cm from the anal verge. At operation, a firm constricting lesion of the rectum was removed with the regional lymph nodes. There was no evidence in either case of disease of the stomach, liver metastasis, or ascites. Pathological studies revealed thickening of the intestinal wall by diffuse infiltrating tumor cells of the characteristic signet cell type and diffuse fibrous tissue throughout the lesion. In the second case, fibrous tissue was especially prominent and cells were very undifferentiated. The most common symptoms of primary linitus plastica were changes in bowel habit (75%), pain and cramps (70%), and blood in stool (gross, 50%; occult, 20%). Characteristic clinical features include a younger age of presentation (average age 56 yr versus 60-70 yr for carcinoma of the colon); a low incidence of hepatic, lung, and bone metastasis (≤15% each); and a high incidence of ovarian (60%) and lymph node-adipose-peritoneal (80%) metastases. The mean duration of survival after diagnosis is only 8.3 months.

8968 RECTAL NECROSIS OF ISCHEMIC ORIGIN: "SPONTANEOUS" AND FOLLOWING AORTO-ILIO-FEMORAL ANGIOPLASTY AND LUMBAR SYMPATHECTOMY. (Fre.) Saegesser, F.; Roenspies, U.; Garcia-Gil, A.;

Boumghar, M. (Service universitaire de chirurgie "A" Chuv, Lausanne, Switzerland). *Chirurgie* 103(10): 907-921; 1977.

Two cases are reported of rectal necrosis attributed to ischemia, and prevention of ischemia of the gastrointestinal tract after infrarenal angioplasty of the iliac aorta is discussed. The first patient, a 62-yr-old man, developed ischemic proctitis with necrosis after an angioplastic operation that involved the aorta and iliac and femoral vessels. However, it is concluded that the ischemia was the result of the low blood flow in pelvic vessels that were obstructed or stenosed. Segmentary resection of the distal part of the sigmoid colon and the upper two-thirds of the rectum was performed on this patient. The surgical specimen showed gangrenous necrosis of the entire gastrointestinal wall with edema and bloody effusion. One year after surgery, rectoscopy showed cyanotic mucosa but no macroscopic ulceration. The second patient, a 68-yr-old man, had necrotic ischemia of the rectal wall as a consequence of repeated episodes of low cardiac output due to paroxysmal tachycardia associated with the Wolff-Parkinson-White syndrome. A recto-sigmoidal resection that included the proximal two-thirds of the rectum was performed. The surgical specimen showed total obliteration of the intestinal mucosa by ulceration and necrosis. Recovery from surgery was satisfactory.

8969 Pseudomyxoma peritonei, CLINICAL AND RADIOGRAPHIC FEATURES. (Eng.) Balthazar, E. J.; Javors, B. R. (Metropolitan Hosp. Center, 1901 First Ave., New York, NY 10029). *Am. J. Gastroenterol.* 68(5):501-509; 1977.

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See also, 8779, 8797, 8802, 8803, 8808, 8819, 8843, 8860, 8926, 8933, 8939, 8945, 9028, 9034, 9037, 9223, 9227, 9230, 9231, 9235, 9238, 9240, 9242, 9247, 9250, 9260.

LARGE INTESTINE

Ulcerative Colitis

9024 CLINICAL OBSERVATIONS ON ULCERATIVE COLITIS. (Eng.) Watanabe, H.; Hiwatashi, N.; Yamagata, S. (Tohoku Univ. Sch. Medicine, Sendai 980, Japan). *Tohoku J. Exp. Med.* 123(3): 197-213; 1977.

The incidence, symptoms, clinical features, complications, treatment, and prognosis of ulcerative colitis were studied in 159 patients. These cases occurred in a total of 102,519 out-patients in Japan between 1954 and 1975, which is 0.15% higher than was believed to occur. The age distribution at the initial examination displayed a bimodal curve in the 79 male patients, with the primary and secondary peaks in the second and sixth decades; there was a unimodal curve in the 80 female patients, with the peak in the fourth decade. A unimodal curve with the peak in the third decade was seen in all patients combined. Of the 94 patients investigated for blood type, 42% were group A, 17% group B, 31% group O, and 10% AB. The precursory symptoms in 109 patients with total and left-sided colitis were mucous blood stools in 81% and abdominal pain in 11%. In 42 patients with proctitis, 83% had mucous blood stools, 17% had diarrhea, and 14% had abdominal pain. A temperature above 37.5 C, a >15% weight loss, diarrhea (>5 stools/day), increased ESR, anemia, leukocytosis (>8,000/mm^3), hypoproteinemia (<6.4 g/100 ml), decreased albumin/globulin ratio (<1.03), and hypopotassemia (<3.5 mEq/l) occurred in 21 of 75, 34 of 73, 50 of 91, 66 of 90, 43 of 85, 34 of 85, 33 of 86, 49 of 82, and 11 of 74 patients with total and left-sided colitis, resp., and in 0 of 27, 3 of 27, 2 of 34, 11 of 28, 6 of 24, 6 of 24, 4 of 30, 7 of 24, and 0 of 19 patients with proctitis, resp. Proctosigmoidoscopy in 79 of 95 patients with total and left-sided colitis in the active stage showed rough or granular mucosa, and erosion was present in 62. These findings were present in 38 of 43 patients with proctitis. Cytologic findings in 95 patients with active total and left-sided colitis included large numbers of neutrophils and the presence of plasma cells in 90 and 67, resp.; these features occurred in 39 and 29 cases, resp., among 43 patients with proctitis. Biopsy specimens from all patients showed goblet cell depletion, crypt abscess, and erosion. Complications in 114 patients with total and left-sided colitis included pseudopolyposis (39), toxic megacolon (3), arthritis (8), stomatitis (3), erythema nodosum (1), and erythema exudativum multiforme (1). Complications in 44 patients with proctitis included pseudopolyposis (6) and arthritis (2). Remission was achieved in 65.5% of the cases followed up. Mortality in these 110 cases was 2.7%. Colectomy was performed in 27.3% of the cases, while ileostomy was carried out in only one case. Among the patients who did not undergo surgery, most were able to return to their occupations.

9025 PRECANCEROUS EPITHELIAL DYSPLASIA IN ULCERATIVE COLITIS: HISTOLOGICAL POSSIBILITY OF EARLY DIAGNOSIS OF COLITIS CARCINOMA. (Ger.) Otto, H. F.; Gebbers, J. O. (Pathologisches Institut der Universitat Hamburg, Martinistrasse 52, D-2000 Hamburg, W. Germany). *Virchows Arch. [Pathol. Anat.]* 377(3):259-276; 1978.

The histology of rectal and colonoscopic biopsy specimens from 20 patients with longstanding colitis (4-22 yr) was studied to determine if precancerous changes are useful for detecting early cancer in colitis. Some patients were monitored by colonoscopy and biopsy for up to 7 yr. A histological study was conducted of rectal and colonoscopic biopsy specimens from 20 patients with colitis of 4- to 20-yr duration. Surgical specimens from a second group of 39 proctocolectomy patients with precancerous epithelial lesions were examined retrospectively; the duration between initial clinical symptoms and proctocolectomy was 3 to 9 yr. Premalignant changes were found in 12 rectal and 14 colonic biopsies from the 20 patients. In the retrospective study, only 3/39 patients were found to have had precancerous lesions. Histologically, the precancerous lesions found among the biopsies could be divided into atypical basal cell proliferation, light-celled epithelial dysplasia, epidermoid metaplasia with atypical cells, adeno-

matous dysplasia, and intermediate forms. Demonstration of severe epithelial dysplasia led to proctocolectomy in 7 of the 20 patients, with an interval of about 2 yr between discovery of dysplasia and surgery. Two of these patients had a carcinoma; the remaining patients were tumor-free. A progression of dysplasia forms could not be demonstrated in any individual, but epidermal metaplasia and atypical basal cell proliferation appeared relatively soon, after 6 and 8 yr of colitis, while adenomatous and villous dysplasia only appeared after a longer period of colitis. Adenocarcinoma always appeared with adenomatous or villous dysplasia, and never with atypical basal cell proliferation. These forms thus present grounds for proctocolectomy.

9026 ANTIBODIES TO SYNTHETIC POLYRIBONUCLEOTIDES IN SPOUSES OF PATIENTS WITH INFLAMMATORY BOWEL DISEASE. (Eng.) DeHoratius, R. J.; Miller, W. C.; Gaeke, R. F.; Strickland, R. G.; Volpicelli, N. A.; Kirsner, J. B.; *et al.* (Dept. Medicine, Univ. New Mexico, Albuquerque, NM 87131). *Lancet* 1(8074):1116-1119; 1978.

To investigate whether RNA viruses are involved in the etiology of inflammatory bowel diseases, sera of 28 patients with Crohn's disease; 16 patients with ulcerative colitis; normal age-, sex-, and race-matched controls; and the unaffected spouses of all subjects were examined for the presence of antibodies to synthetic polyribonucleotides. Significantly more synthetic single-stranded RNA (poly-rA) was bound by serum from patients (230 ng/ml) and from their spouses (185 ng/ml) than by serum from the controls (75 ng/ml, $p<0.002$) or the spouses of the controls (78 ng/ml, $p<0.01$). Similarly, significantly more synthetic double-stranded RNA (poly-rA-poly-rU) was bound by serum from the patients (501 ng/ml) and by serum from their spouses (468 ng/ml) than by serum from the controls (199 ng/ml, $p<0.002$) or by serum from the spouses of the controls (66 ng/ml, $p<0.02$). Binding of poly-rA-poly-rU was similar in patients with Crohn's disease and ulcerative colitis. Poly-rA binding was lower in the patients with ulcerative colitis but was still significantly higher ($p<0.05$) than in matched controls. Binding activity resided in immunoglobulin fractions and was predominantly of IgM class. A significant correlation was observed between the amount of double-stranded but not single-stranded RNA bound by serum from patients and from their respective spouses ($r=0.4$, $p<0.02$). These findings provide indirect support for the presence of RNA viruses in patients with inflammatory bowel diseases and the transmission of such agents to their close personal contacts.

9027 TEN YEARS EXPERIENCE WITH INTRAVENOUS HYPERALIMENTATION AND INFLAMMATORY BOWEL DISEASE. (Eng.) Mullen, J. L.; Hargrove, W. C.; Dudrick, S. J.; Fitts, W. T., Jr.; Rosato, E. F. (Hosp. Univ. Pennsylvania, 3400 Spruce St., Philadelphia, PA 19104). *Ann. Surg.* 187(3):523-529; 1978.

An analysis of parenteral nutritional support provided by i.v. hyperalimentation (IVH) to 74 patients with inflammatory bowel disease (IBD) during 1967-1976 is presented. The patients, 24 with ulcerative colitis (UC) and 50 with granulomatous disease (GD) of the small bowel, colon, or combined disease of both, underwent 117 courses of IVH. On admission for treatment, the patients were malnourished with a weight loss of more than 10% in 63 of them. The malnutrition was of the marasmic-kwashiorkor type with a marked depletion of body-cell mass (weight loss, 19 ± 3 pounds; serum albumin level, 2.9 ± 0.13%; serum transferase level, 158 ± 13.2 mg%). Indications for parenteral nutritional support included nutritional preparation for operation (33%), failure of medical treatment (61%), and the poor nutritional status of the patient (48%). Seventy-seven percent of the patients gained weight on IVH, averaging 10 pounds over the course of treatment (about 3.5 weeks). Almost 40% of the patients who had previously failed on medical regimen had an IVH-induced remission. Therapy before IVH treatment was often of the multiple-drug variety, including steroids, 51%; salicylazosulfapyridine, 51%; and opiates, 57%. After IVH, the amounts of these medications required were 35%, 20%, and 26%, resp. No mortality was attributed to IVH, and complications arose from IVH in only eight patients. Seventeen of 43 enterocutaneous fistulas found in 25 patients were closed with IVH alone. As only three of these recurred, the overall closure rate with IVH alone was 33%. In the surgical ulcerative colitis patients, a correlation was noted between the occurrence of surgical complications and the absence of preoperative steroid treatment. The 10-yr review shows that IVH can ameliorate malnutrition in IBD patients. Combined with complete bowel rest, it can be an effective primary treatment (particularly for GD) or an adjunct to the surgical management of the complications of IBD. Criteria for the use of IVH in IBD are given, and it is noted that, since its efficacy has been documented, nutritional support can be offered to these patients at an earlier stage in the disease.

9028 FILIFORM POLYPOSIS. (Eng.) Zegel, H. G.; Laufer, I. (Hosp. Univ. Pennsylvania, 3400 Spruce St., Philadelphia, PA 19104). *Radiology* 127(3):615-619; 1978.

The cases of four patients with filiform polyposis are presented. This disease was associated with inflammatory bowel disease in three cases, but the fourth patient, a 77-yr-old man, had no prior history of inflammatory disease. The first patient was a 36-yr-old woman who had experienced an episode of ulcerative colitis 2 yr previously but had been in almost complete remission prior to follow-up examination. At this time, a stricture of the transverse colon was found. A subtotal colectomy with ileorectal anastomosis was performed. Pathological examination of the specimen confirmed the presence of a benign stricture and multiple inflammatory polyps, some of which had a long, thin filamentous structure. The second patient was a 20-yr-old woman with granulomatous colitis.

She had been asymptomatic for 4 yr, but a double contrast enema showed several long, filamentous filling defects on a background of normal mucosa in the transverse and descending colon. No further diagnostic or therapeutic procedures were indicated. The third patient, a 33-yr-old man, had an 8-yr history of Crohn's disease involving the small bowel and rectum. He developed epigastric pain, dysphagia, and vomiting, and a double contrast gastroduodenal examination showed deformity of the antrum and pylorus with some loss of circular folds in the proximal duodenum. There was extensive involvement of the distal duodenum and proximal jejunum by Crohn's disease. Numerous, small filamentous filling defects were noted in the body of the stomach. Multiple biopsy specimens showed that these lesions were composed entirely of normal gastric tissue. Biopsies around the margin of a gastric ulcer adjacent to the gastroesophageal junction were compatible with acute gastritis but showed no evidence of granuloma formation. The last patient presented with rectal bleeding, and a filiform polyp of the distal colon was found. Biopsy showed acute and chronic mucosal inflammation, which could indicate either that unrecognized ulcerative colitis or ischemic colitis had been present. It is important to recognize filiform polyposis as a benign, nonspecific sequela of prior, severe inflammatory disease.

9029 CORRELATION OF PLASMA ELECTROLYTES AND FORM ELEMENTS. (Rus.) Revutskii, B. I. (First Dept. Therapy, Donetsk Medical Inst., Donetsk, USSR). *Vrach. Delo* 8:42-44; 1976.

9030 ELECTROLYTE COMPOSITION OF THE BLOOD AND SMALL INTESTINAL SECRETION IN PATIENTS WITH CHRONIC ENTEROCOLITIS RECEIVING DIFFERENT THERAPY. (Rus.) Revutskii, B. I.; Linevskii, Iu. V. (Dept. Therapy, A. M. Gor'kii Donetsk Medical Inst., Donetsk, USSR). *Klin. Med. (Mosk.)* 54 (7):91-95; 1976.

9031 MEDICAL TREATMENT OF HEMORRHAGIC RECTOCOLITIS. (Fre.) Audigier, J. C.; Descos, L. (Hopital Edouard-Herriot, Pavillon H bis, F 69374 Lyon Cedex, France). *Gastroenterol. Clin. Biol.* 1(6/7):577-591; 1977.

9032 THERAPY OF ULCERATIVE COLITIS. (Ger.) Sommer, H.; Kasper, H. (Medizinische Klinik der Universitat, Josef-Schneider-Strasse 2, 8700 Wurzburg, W. Germany). *Dtsch. Med. Wochenschr.* 102(29):1067-1069; 1977.

9033 TREATMENT OF ULCERATIVE COLITIS WITH ASTRONAUT FOOD. (Pol.) Bojanowicz, K. (Klinika Gastroenterologiczna, Instytut Medycyny Wewnetrznej AM, ul. Rewolucji 1905 r. 76 m. 1, 90-223 Lodz, Poland). *Pol. Tyg. Lek.* 32(30):1153-1155; 1977.

9034 USE OF DRY BIFIDUMBACTERIN IN COMPLEX TREATMENT OF CHRONIC INFLAMMATORY INFECTIONS OF THE LARGE INTESTINE. (Rus.) Yukhvidova, Zh. M.; Semenova, L. P.; Kuznetsova, G. C.; Grigoriev, G. A.; Goncharova, G. P. (Central Scientific Res. Inst. Gastroenterology, Moscow, USSR). *Sov. Med.* 2:104-109; 1977.

9035 PRELIMINARY CLINICAL EXPERIENCE WITH A NEW SALAZOSULFAPYRIDINE PREPARATION (COLO-PLEON®) IN THE TREATMENT OF ULCERATIVE COLITIS. (Ger.) Koch, H.; Belohlavek, D. (Medizinische Klinik mit Poliklinik der Universitat Erlangen-Nurnberg, Krankenhausstrasse 12, 8520 Erlangen, W. Germany). *Therapiewoche* 27(29):5331-5332; 1977.

9036 SURGICAL ASPECTS OF ULCERATIVE AND GRANULOMATOUS COLITIS. (Ger.) Stelzner, F. (Chirurgische Universitatsklinik, 53 Bonn-Venusberg, W. Germany). *Therapiewoche* 27(38):6691-6695, *passim*; 1977.

9037 ASSOCIATION OF ULCERATIVE COLITIS, COLONIC CARCINOMA, PLASMACYTOMA AND DERMATITIS HERPETIFORMIS (DUHRING'S DISEASE). (Ger.) Kovary, P. M.; Intorp, H. W.; Lonauer, G.; Losse, H. (Universitats-Hautklinik, V.-Esmarchstrasse 56, 4400 Munster, W. Germany). *Therapiewoche* 26(37):5668-5674, *passim*; 1976.

See also, 8625, 8973, 9231, 9260, 9262.

9038 LIPASE ACTIVITY IN BLOOD FOLLOWING ENDOSCOPIC PANCREATOGRAPHY: DEMONSTRATION OF ITS PANCREATIC ORIGIN AND EXISTENCE OF DUCTAL OR ACINO-VENOUS PATHWAYS IN MAN. (Eng.) Figarella, C.; De Caro, A.; Oi, I.; Sahel, J. (Unite de Recherches de Pathologie Digestive, U 31 Inserm 46, Chemin de la Gaye-13009 Marseille, France). *Scand. J. Gastroenterol.* 13(4):393-399; 1978.

The origin of serum lipolytic activity observed in patients undergoing endoscopic retrograde cholangiopancreatography (ERCP), whether or not followed by nephrography, was investigated. Immediately before and 30 min after cannulation of the Wirsung duct, serum lipase was obtained from 569 patients suspected of pancreatic or biliary disease and who underwent ERCP. The serum lipase properties were compared with those of pancreatic lipase isolated from pancreatic juice obtained by hormonal stimulation of 17 patients before ERCP. Both pancreatic and serum lipase were inhibited by biliary salts above their critical micellar concentrations; the inhibition was reversed by addition of colipase. Studies of serum and pancreatic lipase by chromatography on Sephadex G-100, by electrophoresis, and by double-diffusion immunological assays gave identical results, leading to the conclusion that the serum lipolytic activity in the patients after ERCP was undoubtedly of pancreatic origin. Studies of the patients by post-ERCP nephrography showed an increase in serum lipolytic activity immediately after cannulation of the pancreatic duct and hormonal stimulation and before injection of contrast medium. Activity increased after injection of the dye, leading to pancreatography followed by nephrography, then decreased slowly to a normal value at 40 hr after examination. The presence of pancreatic enzyme activity in serum and the concomitant visualization of the nephron after injection of contrast medium agree with results of previous animal experiments and suggest the presence of acino- or duct-venous pathways in man. In addition to hormonal stimulation, the presence of a tube into the Wirsung duct seems to be necessary to permit an eventual acino- or duct-venous passage. Speculating on the apparent lack of colipase in the serum of certain patients with lipolytic activity and noting that the increased levels of pancreatic enzymes in the ERCP cases are similar to those seen in cases of acute pancreatitis (10-40 times normal), the authors suggest that the estimation of pancreatic enzymes in the blood may be useful in the diagnosis of acute pancreatitis and that the catalytic assay of pancreatic lipase in the blood performed in the presence of an excess of colipase may be a useful test because of its simplicity and rapidity.

9039 BILE ACID METABOLISM IN CYSTIC FIBROSIS. (Eng.) Eklund, A.; Norman, A.; Strandvik, B. *In: Bile Acid Metabolism in Health and Disease. Proceedings of the IV Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-12, 1976.* (Baltimore: University Park Press): 241-246; 1977.

Because cirrhosis and liver damage may occur without the usual liver function tests being abnormal, bile acid metabolism and urinary excretion of bile acids were studied in 15 patients (aged 11 months-18 yr) with cystic fibrosis; all of them had gastrointestinal symptoms and increased fecal fat despite pancreatic enzyme therapy, 14 had pulmonary symptoms, 1 had celiac disease, and 1 was recovering from severe malnutrition due to anorexia nervosa. Liver function tests were normal in all but two patients who showed moderate increases in one or two parameters; BSP retention and/or i.v. galactose elimination were normal in all patients. Of the administered amount of ^{14}C-cholic acid, 13-77% was excreted during the 4-day sampling period; 11-71% of the dose was excreted in the feces; 1-19% of the excreted amount was found in the urine with the percentage increasing significantly with age ($r=0.780$, $p<0.001$). Total urinary bile acid excretion increased with age (from 1 to 27, μmol/day), most of which was due to other than primary bile acids; bile acid concentration (correlated to body weight and square meter body surface) increased in the same way. Bile acids were excreted as free acids (containing most of the trihydroxy bile acids), glycine or taurine conjugates, or sulfated conjugates (containing almost all the monohydroxy bile acids). Despite normal liver function tests, the urinary excretion of isotope after ^{14}C-cholic acid administration increased with age in patients with cystic fibrosis. The urinary bile acid pattern in these patients corresponds to that found in healthy children, but the concentration is higher.

9040 SUBCUTANEOUS CYTOSTEATONECROSIS OF PANCREATIC ORIGIN: SURGICAL PROBLEM IN 5 CASES. (Fre.) Mornet, P.; Guivarc'h, M.; Morin, M.; Marquand, J. (Centre medico-chirurgical Foch, 92151 Suresnes, France). *J. Chir. (Paris)* 114(6): 427-442; 1977.

Five patients with subcutaneous cytosteatonecrosis (Weber-Christian syndrome of pancreatic origin) are reported, and the 68 cases in the world literature are reviewed. The pancreatic pathology was pancreatitis in four patients, only one of whom was an alcoholic, and cancer of the pancreas in one. All five patients had subcutaneous nodules and inflammed painful finger or toe and other joints; abdominal pain and weight loss were experienced by some patients. Elevated levels of amylase in blood and urine and elevated blood lipase level led to suspicion of pancreatic pathology. Abdominal barium transit x-rays showed enlarged duodenum with or without pancreatic calcification in three of five patients. Laparotomy revealed the presence of cancer of the pancreas and hepatic metastases in one patient who had no subsequent treatment. Cystoduodenostomy and duodenopancreatectomy, resp., were performed on two patients with excellent results. The remaining two patients, one of whom was the alcoholic died with widespread cytosteatonecrosis despite cysto jejunostomy and cholecystostomy in the alcoholic and intensive medical treatment with corticosteroids, antienzymes, and aspiration in the other. It is concluded that recovery is possible if early

excision of the affected portion of the pancreas or bypass surgery is performed.

9041 RUPTURED PANCREATIC PSEUDOCYSTS: DIAGNOSIS BY ENDOSCOPY. REPORT OF 3 CASES. (Eng.) Bretholz, A.; Lammli, J.; Corrodi, P.; Knoblauch, M. (Med. Univ.-Klinik, Ramistrasse 100, CH-8091, Zurich, Switzerland). *Endoscopy* 10(1): 19-23; 1978.

A report of three cases of endoscopically proven spontaneous perforation of a pancreatic pseudocyst into the stomach is presented. A 37-yr-old man with a noncontributory history, a 40-yr-old man with a history of multiple bulbar and duodenal ulcers, and a 35-yr-old alcoholic man with a history of acute pancreatitis all presented with acute upper gastrointestinal bleeding. The first patient, who was being treated with anticoagulants for an acute calf-vein thrombosis, had a clinically silent bout of acute pancreatitis (suspected because of an elevation of the amylase level and treated with parenteral nutrition and gastric aspiration) with pseudocyst formation. Endoscopy revealed a large circumscribed protrusion in the posterior wall of the stomach with a hemorrhagic mucosa and giant mucosal folds in the corpus. A 1-cm-wide opening in the fundus developed, and closed slightly over 2 months. The patient was doing well at 1-yr follow-up. The second patient, who had had caudal pancreatic resection for active endocrine adenomata, had perforation of a pancreatic pseudocyst 1 yr later. There was severe diffuse erosive and hemorrhagic gastritis, perforation of the retrogastric process, and a 4-cm opening into a pseudocyst filled with necrotic material. The patient recovered with conservative treatment. In the third patient, bleeding recurred after 2 weeks of conservative management, necessitating laparotomy. An 8-cm pseudocyst in the pancreatic tail and eroding the gastric wall was found. The caudal part of the pancreas and the proximal part of the stomach were resected and an end-to-end esophago-antrostomy performed.

9042 COMPUTED TOMOGRAPHY IN PANCREATIC DISEASE [Abstract]. (Eng.) Fawcitt, R. A.; Isherwood, I.; Braganza, J.; Howat, H. T. (Dept. Diagnostic Radiology, Univ. Manchester, Manchester, England). *Br. J. Radiol.* 51(601):65; 1978.

9043 ULTRASONIC INVESTIGATION OF THE PANCREAS [Abstract]. (Eng.) Berger, L. A. (Royal Free Hosp., London, England). *Br. J. Radiol.* 51 (601):65; 1978.

9044 CIMETIDINE IN TREATMENT OF PANCREATIC INSUFFICIENCY [Letter to Editor]. (Eng.) Porro, G. B.; Dolcini, R.; Grossi, E.; Petrillo, M.; Prada, A. (Ospedale L. Sacco, Milan, Italy). *Lancet* 2(8043):878-879; 1977.

9045 GRANULOMA (SARCOID?) OF THE PANCREAS: A CASE REPORT. (Eng.) Caldwell, J. H.; Evans, W. E. (N-937 Univ. Hosp., Ohio State Univ., 410 W. 10th Ave., Columbus, OH 43210). *Am. J. Gastroenterol.* 69(3, Part 1):320-322; 1978.

9046 CARCINOMA OF THE PANCREAS OCCURRING IN A CHILD: A CASE REPORT WITH DESCRIPTION OF GRAY SCALE ULTRASOUND FINDINGS. (Eng.) Masterson, J. B.; Bowie, J. D.; Port, R. B.; Elahi, C. F.; Burrington, J. D.; Kranzler, J. (Univ. Chicago Hosp. and Clinics, 950 E. 59th St., Chicago, IL 60637). *J. Clin. Ultrasound.*6(3):189-190; 1978.

9047 MECONIUM ILEUS EQUIVALENT. (Eng.) Spiro, R. K.; Landman, M. E. (St. Vincent's Hosp., Montclair, NJ). *Am. J. Gastroenterol.* 69(3, Part 1):302-306; 1978.

9048 CARCINOMA OF THE PANCREAS. (Eng.) Scholz, F. J.; Hatfield, P. M.; Larsen, C. R.; Wise, R. E. (Lahey Clinic Foundation, Boston, MA). *Curr. Probl. Diagn. Radiol.* 7(5):1-41; 1978.

9049 ADENOACANTHOMA OF THE PANCREAS: REPORT OF FOUR CASES AND LITERATURE REVIEW. (Eng.) Weitzner, S. (Univ. Mississippi Medical Center, Jackson, MS 39216). *Am. Surg.* 44(4):206-209; 1978.

9050 CASE REPORT: COMPUTED TOMOGRAPHY OF CYSTADENOMA OF THE PANCREAS. (Eng.) deSantos, L. A.; Bernardino, M. E.; Paulus, D. D.; Martin, R. E. (Univ. Texas System Cancer Center, Houston, TX 77030). *J. Comput. Assist. Tomogr.* 2(2):222-225; 1978.

9051 IS SULPHATED BLOOD-GROUP SUBSTANCE IN PANCREAS A FACTOR IN CYSTIC FIBROSIS? (Eng.) Rolla, G.; Melsen, B.; Sonju, T. (Klinikk for Barnetandpleie og Kariesprofylakse, Geitmyrsveien 71, Oslo 4, Norway). *Acta Pathol. Microbiol. Scand.* [A] 86(1):83-86; 1978.

9052 VISCOSITY OF DUODENAL JUICE EXAMINED IN PATIENTS WITH EXCRETORY PANCREAS INSUFFICIENCY BY THE PANCREOCYMIN [sic] SECRETIN TEST COMPARED TO A CONTROL GROUP AND PATIENTS WITH CHOLELITHIASIS. (Eng.) Leonhardt, H.; Reinhardt, I. (Freie Universitat Berlin, Klinikum Steglitz, D-1000 Berlin 45, W. Germany). *Acta Hepatogastroenterol. (Stuttg.)* 25(1):45-48; 1978.

See also, 8774, 8788, 8789, 8790, 8793, 8795, 8804, 8822, 8879, 8899, 8952, 9063, 9070, 9071, 9178, 9208, 9209, 9212, 9213, 9224, 9225, 9233.

9053 EFFECT OF PROLONGED ADMINISTRATION OF CORTICOSTEROIDS ON EXOCRINE PANCREATIC SECRETION IN MAN. (Fre.) Bourry, J.; Sarles, H. (INSERM U-31, 46, chemin de la Gaye, F 13009 Marseille, France). *Gastroenterol. Clin. Biol.* 2(2): 139-144; 1978.

Because treatment with corticosteroids causes acute pancreatitis in man, exocrine pancreatic secretion was analyzed before and after stimulation with secretin (1 clinical U [CU]/kg) and cholecystokinin-pancreozymin (CCK-PZ, 3 Crick-Harper-Raper U/kg) in 19 patients without pancreatic disease treated with corticosteroids for 46 days to 22 yr. The corticosteroids used in their treatment included betamethasone, triamcinolone, dexamethasone, methylprednisolone, prednisolone, prednisone, hydrocortisone, and tetracosactide. Pancreatic function was compared with that of a control group of 19 sex-, age-, and weight-matched patients without pancreatic disease or steroid treatment. The single sample of pancreatic excretion taken 15 min prior to injection of secretin and CCK-PZ and the two samples taken 15 min and 30 min afterwards were analyzed for lipase, chymotrypsin, phospholipase, bicarbonate, and calcium levels. The absolute concentration of lipase and chymotrypsin and the difference in their concentrations before and after pancreatic stimulation were all significantly ($p<0.05$-0.001) higher in the corticosteroid-treated patients. It is suggested that the hypersecretion of enzymatic proteins in pancreatic fluid of patients on corticosteroids may be the cause of pancreatic lesions.

9054 THE ACTIVITY OF PHOSPHOLIPASE A IN DUODENAL JUICE UNDER PANCREOZYMIN SECRETIN STIMULATION IN CHRONIC PANCREATITIS. (Eng.) Hashihira, S.; Saito, S.; Saeki, M.; Nishii, T.; Takeda, Y.; Mori, R.; *et al.* (Osaka Medical Coll., Takatsuki, Osaka, Japan). *Jpn. Arch. Int. Med.* 24(11): 397-404; 1977.

The cholecystokinin-pancreozymin (CCK-PZ) and secretin tests were carried out in 25 patients with pancreatitis and 18 controls to compare amylase secretion with phospholipase A (PLA) secretion. Eleven patients had calcifying pancreatitis, 5 had definite noncalcifying pancreatitis, and 9 were suspected of having noncalcifying pancreatitis. Controls and patients with pancreatitis showed maximal PLA output 10 min after pancreozymin (1 U/kg body weight, i.v.) stimulation and minimal levels at 40 and 60 min following secretin (1 U/kg body weight, i.v.) stimulation. Stimulated PLA output tended to be lower in the patients with pancreatitis than in the controls, and in the patients with calcifying pancreatitis, this difference was statistically significant ($p<0.05$, 70 min after pancreozymin-secretin stimulation). The amylase output displayed a similar tendency. Although individual deviations were sometimes seen, the total output of both enzymes showed a significant positive correlation ($r=0.837$, $p<0.01$). The PLA/amylase ratios in the patients with calcifying pancreatitis, definite noncalcifying pancreatitis, and suspected noncalcifying pancreatitis, (1.48 ± 0.04, 1.45 ± 0.03, and 1.44 ± 0.03) were significantly higher than those in the controls (1.35 ± 0.02, $p<0.05$). This suggests that PLA maintains its secretory function better than does amylase during pancreatitis.

9055 EARLY OR DELAYED OPERATION IN ACUTE HEMORRHAGIC-NECROTIZING PANCREATITIS? (Eng.) Neher, M.; Mangold, G.; Schonborn, H. (Chirurgische Klinik der Universitat Mainz, W. Germany). *Chir. Gastroenterol.* 11(2):249-252; 1977.

Survival was assessed in patients who underwent early and late operation for necrotizing pancreatitis. Of the 26 patients with acute pancreatitis who underwent early surgery (i.e., after the appearance of necrosis but before the beginning of mortally dangerous complications), 17 had only partial necrosis; 11/17 survived. Of the nine patients who underwent early surgery and had total necrosis, eight survived. Digital removal of necrotic tissue or left-sided resection was possible in the surviving patients. Twenty-three of 32 patients who underwent delayed operation (i.e., after the acute phase) survived. The techniques used were digital removal of sequestric tissue and left-sided resection. The authors conclude that the surgical treatment of total pancreatic necrosis should begin before the onset of lethal complications, but that operation may be delayed for partial pancreatic necrosis, if there are no initial complications or there is no deterioration in the patient's clinical condition.

9056 ALTERATION OF SURFACTANT CHEMISTRY IN EXPERIMENTAL HEMORRHAGIC PANCREATITIS. (Eng.) Maciver, A. G.; Metcalfe, I. L.; Possmayer, F.; Harding, P. G. R.; Passi, R. B. (Univ. Hosp., 339 Windermere Rd., London, Ontario, N6A 5A5 Canada). *J. Surg. Res.* 23(5):311-314; 1977.

The hypothesis that the direct alveolar damage seen in acute pancreatitis may occur secondary to enzymatic degradation of pulmonary surfactant by pancreatic lecithinase was tested in dogs in which hemorrhagic pancreatitis was experimentally induced by transduodenal low pressure injection of autologous bile (0.5 ml/kg) into the main pancreatic duct. Experimental and sham-operated control animals were ventilated for 12 hr and then sacrificed. Saline lavage of the pulmonary alveoli was conducted, and the collected suspension was centrifuged. The lipid was extracted with chloroform; methanol and the phospholipids were separated and assayed by thin-layer chromatography. The total yield of phospholipid was significantly reduced in the experimental group (1.93 ± 0.23 mg/kg

body weight) in comparison with that in controls (4.21 ± 0.24 mg/kg, p<0.005). The lysolecithin fraction was significantly increased in the experimental versus the control group (5.85 ± 0.32% versus 1.31 ± 0.16%, p<0.001). The minimum surface activity of a pulsating bubble in a suspension of the extracted lung wash over 5 min was significantly increased in the experimental dogs (22.4 ± 5.9 dynes/cm) in comparison with that in controls (1.7 ± 0.8 dynes/cm, p<0.01). These results demonstrate that the recovery of surfactant is reduced and that the percentage of lysolecithin is increased in hemorrhagic pancreatitis, which supports the stated hypothesis.

9057 PANCREATITIS FOLLOWING KIDNEY TRANSPLANTATION. (Ger.) Scholzel, E.; Largiader, F.; Uhlschmid, G.; Binswanger, U.; Corrodi, P.; Knoblauch, M. (Chirurgische Universitatsklinik A, Zurich, Switzerland). *Schweiz. Med. Wochenschr.* 108(5):161-165; 1978.

Pancreatitis following kidney transplant presents a serious, often life-threatening complication. Follow-up of 249 recipients of cadaver renal allografts showed that 14 had contracted pancreatitis 1 week to 7 yr and 4 months following transplantation. Five patients died as a direct result of the disease, while pancreatitis discovered at autopsy was implicated in the death of two patients. The incidence of pancreatitis was 5.6%, with a mortality rate of 50%; participation of pancreatitis in death was 7%. Etiologic factors in this complication included pancreatic damage caused by chronic uremia before the transplant, treatment with L-asparaginase, and damage from infections. Pancreatitis following kidney transplantation is thus thought to be of multifactorial origin, with steroid therapy predominant in causing pancreatic damage.

9058 PANCREATITIS FOLLOWING SPINAL CORD INJURY. (Eng.) Carey, M. E.; Nance, F. C.; Kirgis, H. D.; Young, H. F.; Megison, L. C., Jr.; Kline, D. G. (Louisiana State Univ. Medical Center, 1542 Tulane Ave., New Orleans, LA 70112). *J. Neurosurg.* 47(6):917-922; 1977.

Six cases of pancreatitis following spinal cord injury in otherwise healthy individuals are reported. Four patients manifested signs and symptoms of overt pancreatitis having variously, fever, protracted nausea, vomiting, or epigastric tenderness. Two patients died from the effects of grossly or microscopically confirmed acute hemorrhagic pancreatitis. All patients had ileus because of their cord lesion. None had hyperparathyroidism, aminoaciduria, hyperlipemia, hemochromatosis, or cholelithiasis, and none was alcoholic. Only one patient may have sustained pancreatic trauma, and only one was hypothermic. The possibility of penetrating ulcer was excluded in four cases. None of the patients had prolonged steroid therapy; one received no steroids and another received only a single 12-mg injection of dexamethasone. No single, etiologically accepted mechanism already postulated to cause pancreatitis can account for all the cases reported. The authors hypothesize that spinal cord disruption may produce pancreatitis by sympathetic-parasympathetic nervous system imbalance resulting in over-stimulation of the sphincter of Oddi. This may lead to stasis of secretions with absorption of amylase into the systemic circulation, and structural pancreatic damage. Pancreatitis in patients with cord injuries is easily overlooked because abdominal pain is usually absent and fever is usually attributed to more frequently occurring pulmonary or urinary tract infections. Recognition of this complication is important to decrease the morbidity and mortality mortality that follows spinal cord damage.

9059 ACUTE PANCREATITIS: THE QUEENSLAND SCENE. (Eng.) Battersby, C.; Chapuis, P. (Princess Alexandra Hosp., Ipswich Rd., Woolloongabba, Queensland 4102, Australia). *Aust. N.Z. J. Surg.* 47(2):204-209; 1977.

9060 IATROGENIC PANCREATITIS. (Eng.) Anonymous (No affiliation given). *Br. Med. J.* 2(6094):1043; 1977.

9061 PREDICTIVE VALUE OF BIOCHEMICAL INDEXES IN ACUTE ALCOHOLIC PANCREATITIS. (Eng.) Palani, C. K.; Sehgal, L. R.; Kraft, A. R.; Duarte, B.; Dulski, R.; Levine, H.; Moss, G. S. (Cook County Hosp., Chicago, IL). *Surg. Forum* 28:441-443; 1977.

9062 SPONTANEOUS DRAINAGE OF A PANCREATIC CYST WITHOUT REFILL. (Eng.) Raymond, S. W.; Hemphill, R. B. (West Suburban Hosp., Oak Park, IL). *Abdom. Surg.* 19(6):143-146; 1977.

9063 ENDOSCOPIC CANNULATION OF THE PAPILLA OF VATER. (Eng.) Cotton, P. B. (Middlesex Hosp., London W1, England). *Rend. Gastroenterol.* 9(2):144-149; 1977.

9064 NUTRITIONAL HABITS OF ACUTE PANCREATITIS PATIENTS IN VERONA, ITALY. (Eng.) Angelini, G.; Cavallini, G.; Vantini, I.; Rosa, B.; Adamo, S.; Zuniga, G.; *et al.* (Clinica Medica III, Universita di Padova, Sede di Verona, 37100 Verona, Italy). *Rend. Gastroenterol.* 9(2):114-117; 1977.

9065 ACUTE PANCREATITIS. (Fre.) Boutelier, Ph. (No affiliation given). *Chirurgie* 103(9):856-857; 1977.

9066 PANCREATITIS DUE TO OBSTRUCTION OF ODDI'S SPHINCTER. (Eng.) Mouiel, J.; Bourgeon, R.; Chauvin, P.; Bertrand, J. C.; Giaume, F.; Rey,

J. F. *In: The Sphincter of Oddi. Proceedings of the Third Gastroenterological Symposium, Nice, June 8-9, 1976.* (Basel: S. Karger): 163-174; 1977.

9067 THE PART THAT THE SPHINCTER OF ODDI PLAYS IN THE ETIOLOGY OF PANCREATITIS. (Eng.) White, T. T. *In: The Sphincter of Oddi. Proceedings of the Third Gastroenterological Symposium, Nice, June 8-9, 1976.* (Basel: S. Karger): 175-179; 1977.

9068 NEWER CONCEPT FOR THE PATHOGENESIS OF CHRONIC ALCOHOLIC PANCREATITIS. (Eng.) Bordalo, O.; Goncalves, D.; Noronha, M.; Cristina, M. L.; Salgadinho, A.; Dreiling, D. A. (Faculty Medicine, Lisbon, Portugal). *Am. J. Gastroenterol.* 68(3):278-285; 1977.

9069 PANCREATIC PLEUROPERICARDIAL EFFUSIONS PRESENTING AS TUMOR OF THE LUNG. (Eng.) Ploy-Song-Sang, Y.; Vassallo, C. L. (Univ. Cincinnati Medical Center, Room 7510, Medical Sciences Building, 231 Bethesda Ave., Cincinnati, OH 45267). *South. Med. J.* 70(12):1474-1476; 1977.

9070 CHRONIC PANCREATIC ASCITES AND PANCREATIC PLEURAL EFFUSIONS. (Eng.) Cameron, J. L. (Johns Hopkins Medical Inst., Baltimore, MD 21205). *Gastroenterology* 74(1):134-140; 1978.

9071 CHRONIC PANCREATITIS IN ITALY. AETIOLOGICAL, CLINICAL AND HISTOLOGICAL OBSERVATIONS BASED ON 253 CASES. (Eng.) Gullo, L.; Costa, P. L.; Labo, G. (Policlinico S. Orsola, Via Massarenti, 9, 40138 Bologna, Italy). *Rend. Gastroenterol.* 9(2): 97-104; 1977.

9072 INTERPRETATION OF AMYLASE CLEARANCE IN PATIENTS WITH ABNORMAL CREATININE CLEARANCE. (Eng.) Payne, R. B. (St. James' Univ. Hosp., Leeds LS9 7TF, England). *Br. Med. J.* 1(6104):22; 1978.

9073 PANCREATIC STIMULATION WITH GIH AND BOOTS SECRETIN IN NORMAL AND CHRONIC PANCREATITIC [sic] SUBJECTS. (Eng.) Bardella, M. T.; Terruzzi, V.; Ranzi, T.; Bianchi, P. A. (First Dept. Medicine, Univ. Milan, Milan, Italy). *Panminerva Med.* 19(5): 295-298; 1977.

9074 SWEAT ELECTROLYTES IN CHRONIC PANCREATITIS. (Eng.) Bank, S.; Marks, I. N.; Novis, B. (Long Island Jewish Hosp., New Hyde Park, NY). *Am. J. Dig. Dis.* 23(2):178-181; 1978.

9075 THE TIMING OF CHOLECYSTECTOMY IN PATIENTS WITH GALLSTONE PANCREATITIS. (Eng.) Yague, S. (San Juan de la Cruz Hosp., Ubeda, Jaen, Spain). *Abdom. Surg.* 20(2):27-29; 1978.

9076 SYMPOSIUM ON PANCREATITIS: 1. CONSERVATIVE MANAGEMENT OF ACUTE PANCREATITIS. (Eng.) Keith, R. G. (Sunnybrook Medical Center, 2075 Bayview Ave., Toronto, Ontario M4N 3M5, Canada). *Can. J. Surg.* 21(1):56-58; 1978.

9077 SYMPOSIUM ON PANCREATITIS: 2. SURGICAL MANAGEMENT OF ACUTE PANCREATITIS. (Eng.) Poncelet, P. (Centre Hospitalier de l'Universite Laval, 2705 boul. Laurier, Quebec G1V 4G2, Canada). *Can. J. Surg.* 21(1):58-59; 1978.

9078 SYMPOSIUM ON PANCREATITIS: 4. SURGERY IN CHRONIC PANCREATITIS. (Eng.) Turner, F. W. (Dept. Surgery, Univ. Alberta, Edmonton, Alberta, Canada). *Can. J. Surg.* 21(1):63-67; 1978.

See also, 8704, 8780, 8907, 8916, 8952, 9040, 9042, 9043.

9079 AUTOMATIC ANALYSIS OF BSP KINETICS IN THE NORMAL INDIVIDUAL AND IN LIVER PATIENTS. (Ita.) Mattei, M.; Avanzolini, G.; Gnudi, G.; Malaguti, P.; Sciarretta, G.; Ruscelli, C.; *et al.* (Ospedale Maggiore "C. A. Pizzardi," Bologna, Italy). *Minerva Dietol. Gastroenterol.* 23(2):79-83; 1977.

To gain greater clinical information from the BSP elimination test, injections of 5 mg/kg of BSP in a 5% solution were given to 10 healthy individuals, 41 with nonhepatic digestive disease, 10 with steatosis, 10 with cirrhosis, 9 with chronic active hepatitis, 6 with granulomatous hepatopathy, 3 with cancer and cirrhosis, and 3 with metastases. Blood samples were taken at various time periods ranging from 4 to 120 min after the injection. The retention of BSP after 45 min was 1.9 ± 0.5% in the control group, 3.9 ± 2% in those with nonhepatic digestive disease, 12.5 ± 6.6% in those with steatosis, 24.6 ± 6.5% in those with cirrhosis, 17.8 ± 8% in those with chronic active hepatitis, 9.5 ± 6.6% in those with granulomatous hepatopathy, 11 ± 6% in those with cancer and cirrhosis, and 18 ± 11.3% in those with metastases. A two-compartment (serum and liver) mathematical analysis of the decrease in BSP levels was made and the coefficients of exchange were determined. It is concluded that the mathematical model is more discriminating in patients with hepatopathy than with other types of disease.

9080 ELECTROMYOGRAPHY IN THE DETECTION OF PERIPHERAL NEUROPATHY IN CHRONIC LIVER DISEASE. (Ita.) Alvisi, V.; Degli Uberti, E.; Tralli, G. (Istituto di Patologia Speciale Medica e Metodologia Clinica, Universita degli Studi di Ferrara, Ferrara, Italy). *Minerva Med.* 68(62): 4183-4191; 1977.

Electromyographic studies were done on 29 patients who had had hepatopathy for not less than 3 yr (diabetics and alcoholics were excluded). The neurologic examination revealed neuropathy (paresthesia, hypoesthesia, and osteotendinous hypo-areflexia) in 9/29 patients. Among the 13 with chronic latent hepatopathy, 4 showed neuropathy, while among the 16 with overt chronic hepatopathy, 5 had neuropathy. The velocity of minimum motor conduction (VC MIN) of the ulnar nerve was significantly slower in patients with chronic latent hepatopathy with clinical signs of neuropathy (47.7 m/sec, $p<0.05$) than in healthy controls. In these patients, both the VC MIN and the velocity of maximum conduction (VC MAX) for the external poplitic sciatic nerve were significantly slower (37.75 m/sec and 43.875 m/sec, resp., $p<0.05$) than in the controls. In patients with overt chronic hepatopathy without clinical signs of neuropathy, the VC MAX (54.476 m/sec) and VC MIN (48.585 m/sec) of the ulnar nerve were significantly lower ($p<0.01$) than in the controls. In these patients the VC MAX (44.904 m/sec), VC MIN 37.985 m/sec), and the dispersion (6.78 m/sec) were all lower ($p<0.001$) than in the controls. In patients with overt chronic hepatopathy showing clinical signs of neuropathy, the VC MIN (46.20 m/sec) and the VC MAX (53.34 m/sec) were significantly lower in the ulnar nerve ($p<0.01$) and the VC MAX (43.06 m/sec), the VC MIN (33.966 m/sec), and the dispersion (8.8 m/sec) were significantly lower ($p<0.001$) for the external poplitic sciatic nerve. The results confirm that in cases of chronic hepatopathy neuropathy can develop.

9081 SKIN REACTIONS CAUSED BY VITAMIN K IN PATIENTS WITH LIVER DISEASE. (Eng.) Bullen, A. W.; Miller, J. P.; Cunliffe, W. J.; Losowsky, M. S. (St. James' Hosp., Leeds LS9 7TF, England). *Br. J. Dermatol.* 98(5):561-565; 1978.

Six cases are reported in which patients with chronic liver disease developed cutaneous reactions around the site of injection of oil-soluble vitamin K. The erythematous rash appeared at the injection site 7-16 days after the initiation of vitamin K therapy for a raised prothrombin time. In four patients, the rash spread, either towards the sacrum or more frequently, down to the thigh. Resolution occurred with scaling with 9-22 days. The amount of vitamin K given before the onset of the rash varied between 270 and 440 mg. Five patients subsequently received a water-soluble vitamin K analogue with no adverse affects. In two patients skin tested by the intradermal injection of 0.1 ml of the vitamin K preparation within 1 week of the development of the rash, induration and erythema occurred. This reaction was maximal at 24-72 hr. Pruritus occurred at the site of injection from 24 hr to 15 days after the skin test. There was no significant skin reaction in these two patients when menadiol sodium diphosphate or the different components of the oil-soluble preparation of vitamin K were injected. Similar results were obtained in three other patients tested several months after the development of the initial reaction. Lymphocyte transformation was not stimulated by up to 200 µg/ml of the preparation, nor was leukocyte migration inhibited by 500 µg/ml in the one patient tested. Skin biopsy of the lesion showed a nonspecific perivascular infiltration with chronic inflammatory cells in the dermis. Staining for IgG, IgA, IgM, IgE, and the C_3 component of complement gave negative results. Skin testing was also carried out in 10 other patients with chronic liver disease, none of whom had developed a skin reaction to i.m. vitamin, and in 10 healthy controls. Four patients with liver disease and two healthy volunteers developed a reaction to intradermal vitamin K (0.1 ml). The pathogenesis of this reaction remains uncertain, but the authors suggest that large doses of vitamin K are implicated, and that the reaction may not be immunologically determined.

9082 DOES FATTY LIVER INDUCED BY PARENTERAL NUTRITION DEPEND ON THE AMOUNT OF GLUCOSE SUPPLIED? (Fre.) Messing, B.; Bitoun, A.; Galian, A.; Mary, J. Y.; Goll, A.; Bernier, J. J. (Hopital Saint-Lazare, 107 bis, rue du Faubourg-Saint-Denis, F 75010 Paris, France). *Gastroenterol. Clin. Biol.* 1(12):1015-1025; 1977.

A retrospective study of 15 patients fed parenterally for 7-500 days with high protein (0.20-0.55 g N/kg/24 hr) and variable glucose (40-100 kcal/kg/24 hr) showed 4 patients who definitely developed fatty liver and 5 suspected cases. Criteria for selection of the patients for study were (1) absence of hyperlipemia prior to parenteral feeding (triglycerides $\leq$1.50 g/l, total cholesterol $\leq$2.60 g/l), (2) absence of evidence of liver disease, (3) absence of diabetes, (4) precisely defined nutrition, and (5) weekly evaluation of liver function and blood lipids. The nine patients with confirmed or suspected fatty liver received significantly ($p<0.015$) higher levels of glucose (68.89 ± 6.05 kcal/kg/24 hr) than did the other six patients (45.83 ± 3.52 kcal/kg/24 hr). In addition, these nine patients had hypertriglyceridemia (1.60-7.60 g/l) with an average increase in triglycerides of 3.27 ± 0.69 g/l compared with an increase of 0.37 ± 0.07 g/l ($p<0.005$) in the six patients without fatty liver. The presence of fatty liver was determined by biopsy and by measurement of the levels of transaminases and alkaline phosphatase; hepatomegaly developed in five patients and icterus in two. It is concluded that glucose should not be excessive in parenteral nutrition, and when necessary to supply caloric needs, fatty acids should be substituted.

9083 HEPATIC HEMANGIOMAS: PITFALLS IN SCINTIGRAPHIC DETECTION. (Eng.) Good, L. I.; Alavi, A.; Trotman, B. W.; Oleaga, J. A.; Eymontt, M. J. (Hosp. Univ. Pennsylvania, 3600 Spruce St., Philadelphia, PA 19104). *Gastroenterology* 74(4): 752-758; 1978.

Recent experiences with three patients with angiographically proven hepatic hemangiomas that were not always detected by radionuclide blood flow and blood pool scintigrams of the liver are presented. In the first patient, hepatomegaly associated with a large solitary scintigraphic defect of the liver was present. A rapid-sequence flow study after the i.v. injection of 15 mCi of ^{99m}Tc-pertechnetate revealed no flow to the large defect during the 1 min that images were obtained. Ultrasound examination revealed an inhomogeneous echo pattern in the right half of the liver, which suggested a large solid intrahepatic mass. A liver biopsy produced hepatic bleeding, and at laparotomy an extensive cavernous hemangioma was found. Postoperatively, a hepatic arteriogram revealed a massive cavernous hemangioma involving the entire liver. At a second laparotomy, the right hepatic artery was ligated; 1 yr later, a blood pool image revealed a pattern consistent with a hemangioma. The second patient had hepatomegaly with a scintigraphic space-occupying mass in the right lobe. A rapid-sequence flow study revealed no appreciable uptake of the radionuclide in the defect, and a blood pool image showed a smaller defect in the same location, suggesting vascularity in the periphery of the lesion. Hepatic angiography showed a large hemangioma in the right lobe and many other small ones scattered throughout both lobes. The third patient did not have an enlarged liver, but liver scans revealed a solitary defect in the right lobe. On hepatic blood flow and blood pool scans, this lesion appeared to have less activity than did the surrounding hepatic parenchyma. A hepatic arteriogram revealed a solitary circumscribed hemangioma in the posterior aspect of the right lobe. On the basis of this experience, the authors recommend hepatic angiography for definitive evaluation of the vascularity of solitary defects found on routine liver scans.

9084 LYSOSOMAL DISRUPTION IN THE PATHOGENESIS OF HEPATIC DAMAGE IN PRIMARY AND SECONDARY HAEMOCHROMATOSIS. (Eng.) Peters, T. J.; Selden, C.; Seymour, C. A. (Royal Postgraduate Medical Sch., London, England). *CIBA Found. Symp.* 51(New Series):317-329; 1977.

Evidence that lysosomes accumulate iron in the liver cells of patients with hemochromatosis and that this accumulation is implicated in the pathogenesis of the tissue damage is reviewed. Morphology of liver, spleen, myocardium, bone marrow cells, and enterocytes from patients with iron overload demonstrated prominent lysosomes, presumed to contain iron compounds. The activities of two lysosomal enzymes, acid phosphatase and *N*-acetyl-β-glucosaminidase, were significantly elevated in liver biopsies from patients with primary or secondary hemochromatosis, which was consistent with the intralysosomal accumulation of iron. Subcellular fractionation by sucrose density gradient centrifugation of liver biopsy specimens from a patient undergoing venesection for hemochromatosis showed that at least 85% of both *N*-acetyl-β-glucosaminidase and β-galactosidase activities were present as nonsedimentable enzyme remaining in the sample layer. After venesection, the iron content was about one-third of that in untreated tissue but was still six times greater than that of control tissue. About half the enzyme activity remained in the sample layer, while the remainder was associated with lysosomes. However, the density distribution of the lysosomes was strikingly abnormal, with an equilibrium density of 1.29 compared with 1.20 in control tissue. These high-density lysosomes had a unique enzyme composition containing significant amounts of *N*-acetyl-β-glucosaminidase and β-galactosidase activity but relatively little acid phosphatase and β-glucuronidase activity. Biopsy extracts after further venesection showed normal tissue iron concentrations and normal acid hydrolase distribution. Electron microscopy of tissue before treatment showed distended hemosiderin-containing lysosomes. Iron-laden lysosomes have also been observed in iron-overloaded rats. Increased activities of acid hydrolases and enhanced lysosomal fragility in liver homogenates have been demonstrated in patients but not in experimental animals.

9085 GENETICALLY DETERMINED IMMUNE HYPERREACTIVITY IN HUMAN LIVER DISEASE. (Eng.) Eddleston, A. L. W. F. (King's Coll. Hosp., London SE5, England). *Proc. R. Soc. Med.* 70(8):525-529; 1977.

Evidence suggesting that there is an inherited immune hyper-reactivity in many patients with chronic active hepatitis (CAH) is reviewed. Family studies have shown that many of the first-degree relatives of patients with hepatitis B surface antigen (HBsAg)-negative CAH have raised serum gamma-globulin levels in the absence of any clinical or biochemical signs of liver disease; this is compelling evidence against the hypothesis that the hypergamma-globulinemia in these patients is entirely secondary to liver damage. In addition, over 60% of these patients have been found to possess the same histocompatibility antigen, HLA-B8. In patients with HLA-B8 and HBsAg-negative CAH, the mean titers of antibodies to rubella, measles, smooth muscle, nuclei, and *Escherichia coli* have been found to be higher than in patients without the histocompatibility antigen; in the case of rubella antibodies, the difference is significant. Among relatives of all these patients with chronic hepatitis, the mean titer of rubella antibodies was found to be significantly higher in the first-degree relatives than in the genetically-unrelated spouses, but these high antibody levels were not related to the presence of HLA-B8. This is evidence against the proposed simple inheritance of an immune-response gene linked to HLA-B8, but is consistent with the inheritance of a more generalized nonspecific increase in immune responsiveness that is under polygenic control. One author has suggested that a defect in the activity of suppressor T cells may be of central importance in the persistence of autoimmune reactions and thus the pathogenesis of autoimmune tissue injury. The present author has postulated that HLA-B8 might be a marker for such an abnormality in the control of immune responses and that this defect could be the link between the many different HLA-B8 associated diseases. A suppressor T-cell defect would result in a failure to switch off immune responses adequately, but only when combined with a powerful switch-on mechanism would it lead to very high antibody levels. The parallelism between this predicted behavior and the actual findings in patients with CAH and their relatives is remarkably close.

9086 SECONDARY HYPERTRIGLYCERIDAEMIA IN PATIENTS WITH PARENCHYMAL LIVER DISEASE. (Eng.) Klose, G.; Windelband, J.; Weizel, A.; Greten, H. (Klinisches Institut fur Herzinfarktforschung an der Medizinischen Universitatsklinik, Bergheimer Strasse 58, D-69 Heidelberg, W. Germany). *Eur. J. Clin. Invest.* 7(6):557-562; 1977.

The role of hepatic triglyceride lipase (H-TGL) and plasma lipoprotein lipase (LPL) in liver diseases was studied by selective precipitation of H-TGL with specific antibodies and subsequent measurement of both activities. The subjects included 50 patients with acute hepatitis, 20 patients with chronic active or persistent hepatitis, 50 patients with liver cirrhosis, and 47 healthy volunteers. All enzyme activities are in units of log μmol x 100 free fatty acids released 1 ml/hr. The mean H-TGL activity in the 22 normal men was 38.94 ± 4.54, and that in the 25 normal women was 36.66 ± 5.94. The mean levels in the 26 women with acute hepatitis (30.91 ± 8.64, $p<0.05$), the 24 men with acute hepatitis (31.06 ± 20.0, $p<0.05$), the 14 women with chronic hepatitis (29.90 ± 18.80, $p<0.05$), the 13 women with liver cirrhosis (28.40 ± 19.0, $p<0.01$), and the 37 men with liver cirrhosis (29.0 ± 13.8, $p<0.01$) were significantly lower than the corresponding values in the normal men and women. The mean LPL level in women with acute hepatitis (27.40 ± 18.5) was significantly lower ($p<0.01$) than the LPL levels in normal men or women (34.36 ± 4.44 and 32.27 ± 4.55, resp.), but all other levels were similar to the control levels. The possibility of a circulating inhibitor in the patient's serum was ruled out by mixing enzyme preparations from normal controls with plasma from liver patients. Both H-TGL and LPL were not inhibited with increasing amounts of such serum in the incubation medium. The LPL level did not correlate significantly with any liver function test. There were significant correlations between H-TGL activity and the levels of plasma triglycerides ($r=0.3002$, $p<0.05$), alkaline phosphatase ($r=0.4350$, $p<0.01$), and lecithin-cholesterol-acyltransferase (LCAT, ($r=0.2994$, $p<0.05$) in acute hepatitis; between H-TGL activity and SGPT level ($r=0.4836$, $p<0.05$) and between H-TGL and SGOT level ($r=0.4521$, $p<0.05$) in chronic hepatitis; and between H-TGL activity and serum bilirubin level ($r=0.4463$, $p<0.01$), between H-TGL activity and the normotest ($r=0.5966$, $p<0.01$), and between H-TGL and LCAT activities ($r=0.5965$, $p<0.01$) in liver cirrhosis. Total post-heparin lipolytic activity decreased with the severity of liver dysfunction. With improvement, hypertriglyceridemia disappeared with a concomitant increase in the H-TGL and LPL levels. It is concluded that impaired triglyceride metabolism in liver disease is at least partly caused by diminished plasma hepatic TGL activity.

9087 SONOGRAPHY AND CONVENTIONAL RADIOGRAPHY FOR POSTOPERATIVE LOCAL INFECTIONS IN THE UPPER ABDOMEN. (Ger.) Vogel, H.; Scherer, K.; Eckert, P.; Vogel-Karl, B. (Radiologische Klinik, Universitatskrankenhaus Hamburg-Eppendorf, 2000 Hamburg 20, W. Germany). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 127(5):433-439; 1977.

Sonographic and conventional radiographic examinations were performed on 24 patients with suspected postoperative abscesses and/or empyemas of the upper abdomen. Total or partial pancreatectomy was performed in 5 cases, Billroth I antireflux plastic surgery and other interventions in the upper digestive tract in 12, surgery on the hepatobiliary system in 5, and combined surgery in 2. Thirty-two abscesses and empyemas were subsequently diagnosed by puncture and reoperation. Sonography demonstrated all 8 liver abscesses, 11/21 abdominal empyemas, and 3 abscesses or empyemas in other positions. Radiography demonstrated 5/8 liver abscesses, 16/21 upper abdominal empyemas, and 1 of the remaining lesions. Sonography demonstrated fluid-filled cavities, while radiography showed especially the presence of extraluminal air. The two methods are consequently complementary. Sonography should be the first diagnostic method

used whenever postoperative liver abscess is suspected, while radiography should precede sonography in case of suspected postoperative empyema.

9088 HEPATIC FINDINGS IN JUVENILE CHRONIC POLYARTHRITIS. (Ger.) Rau, R. (Stadtspital Triemli, CH-8063 Zurich, Switzerland). *Schweiz. Med. Wochenschr.* 107(35):1235-1237; 1977.

Hepatic findings in patients with juvenile chronic polyarthritis (JCP) and Chauffard's syndrome (CS) are reviewed. Hepatomegaly was found in 2.9% of patients with JCP as strictly defined, in 48% of the patients with incomplete CS, and in 79% of the patients with complete CS. This hepatomegaly increased during recurrences, and disappeared during remissions. Transient jaundice, elevation of amino transferase levels, and delayed BSP excretion were reported, particularly in patients with complete CS; these findings are indicative of impaired liver function. Liver biopsies have been performed only rarely; they demonstrated nonspecific lymphocyte infiltration of portal fields and, in a few cases, autoimmune hepatitis and even liver cirrhosis with portal hypertension. Plasma cell hepatitis with arthropathy can be easily differentiated from juvenile rheumatoid arthritis: in the former, the synovitis is merely transient and disappears under steroid therapy. As in adults, severe liver dysfunction leads to remission of arthritis. Amyloidosis should be considered in every case of long-lasting hepatomegaly.

9089 IMMUNOGLOBULINS IN SERA OF PATIENTS WITH PORPHYRIA CUTANEA TARDA. (Eng.) Kordac, V.; Fucikova, T. (Charles Univ. Prague, Dept. Clinical Immunology, Unemocnice 2, 12808 Praha 2, Czechoslovakia). *Acta Hepatogastroenterol. (Stuttg.)* 25(1):20-22; 1978.

The serum immunoglobulins were investigated in 67 patients with porphyria cutanea tarda (PCT) and compared with those in 163 healthy controls and 37 age- and sex-matched controls. The typical laboratory findings of PCT were confirmed in these patients (increased BSP retention and SGPT level, positive thymol turbidity reaction). IgG and IgM levels were significantly lower in PCT patients without liver cirrhosis (both those not treated, the S group, and those treated with venepunctures, the V group) than in the controls. The IgG and IgM levels were 150 and 175 IU, resp., in controls, 125 IU ($p<0.05$) and 138 IU ($p<0.01$), resp., in the S group, and 142 IU ($p<0.01$) and 163 IU ($p<0.01$), resp., in the V group. The IgA levels in all patients and the IgG and IgM levels in the untreated PCT patients with cirrhosis were not significantly different from control values. The authors suggest that some PCT patients may have an altered humoral immunity.

9090 ERYTHROPOIETIC PROTOPORPHYRIA: JUVENILE PROTOPORPHYRIN HEPATOPATHY CIRRHOSIS AND DEATH. (Eng.) Cripps, D. J.; Gilbert, L. A.; Goldfarb, S. S. (Univ. Wisconsin Sch. Medicine, 1300 University Ave., Madison, WI 53706). *J. Pediatr.* 91(5):744-748; 1977.

The case of an 11-yr-old boy with erythropoietic protoporphyria complicated by severe hepatic cirrhosis and failure is described. The patient had a 10-yr history of photosensitivity. He presented with jaundice, fever, acute dermatitis, edema, and tiredness. Hepatosplenomegaly, ascites, and periumbilical vein distention were present. The patient subsequently developed a gastric ulcer with perforation, and liver function deteriorated. Episodes of hematemesis, due to esophageal varices, occurred, and the patient died. The terminal erythrocyte protoporphyrin values were 8,650 to 9,650 µg/dl. Stool protoporphyrin was 1,580 µg/g dry weight, and the estimated level in the liver was 57.5 mg/g wet weight. At autopsy, the liver showed typical features of postnecrotic cirrhosis, was greatly enlarged (1,700 g), and distorted by numerous hyperplastic nodules. Masson trichrome stain indicated dense fibrous scars separating hepatocellular nodules. The hyperplastic nodules contained hepatocytes and Kupffer cells containing clumps of brown pigment. When examined by fluorescence microscopy with a quartz iodine tungsten light, the clumps emitted a persisting red fluorescence, and when examined under polarized light, appeared birefringent with prominent Maltese-cross patterns. Ultrastructural examination showed distinct aggregates of crystalline structures. They were composed of short, straight, or slightly curved barlike structures with tapered ends. Lymph nodes in the region of the porta hepatis and around the head of the pancreas contained abundant doubly refractile pigment similar to that of the liver parenchyma. Although severe liver damage in erythropoietic protoporphyria is rare, patients with this disease should be followed regularly with liver function tests, and a liver biopsy at the time of initial diagnosis may be considered.

9091 FERRITIN IN HUMAN LIVER CELLS OF HOMOZYGOUS BETA-THALASSAEMIA: ULTRASTRUCTURAL OBSERVATIONS. (Eng.) Iancu, T. C.; Neustein, H. B. (Lady Davis Carmel Hosp., Haifa, Israel). *Br. J. Haematol.* 37(4):527-535; 1977.

Iron overload was studied ultrastructurally in 16 biopsy specimens from 10 patients, of whom 9 had homozygous beta-thalassemia and 1 had delta-beta-thalassemia. A number of round or oval lysosomal structures containing lamellae different from myelin figures were seen in all patients, including those with minimal iron overload. Ferritin molecules were seen either forming arrays in relation with the lamellae, or in paracrystalline arrangement, or with no organized form. There were practically no ferritin molecules in subcellular compartments other than cell sap and lysosomes. The concentration of randomly dispersed ferritin molecules in the cell sap of hepatocytes in the three patients under 13 months of age was noticeably lower than that in older patients; the mean number of ferritin particles in the former patients were 0.7, 0.5, and 0.1 particles/cm^2. In patients over

3 yr of age and up to 22 yr, the cell sap counts were 4.1-10.6 particles/cm^2, and the counts were unrelated to age. Hemosiderin was found only in lysosomes and had various densities. Hemosiderin-containing lysosomes often contained lipid droplets and/or myelin figures. Compared to hepatocytes, the Kupffer cells had a higher concentration of ferritin in the cell sap, the number ranging between 20.8 to 30.6/cm^2 in patients older than 3 yr. In younger patients, the number of particles was generally lower, but were variable from cell to cell. Kupffer cells also showed a large number of iron-containing lysosomes. These observations reveal that ferritin molecules vary in dimension, arrangement, distribution, and aggregation within subcellular compartments in prolonged iron overload. The round or oval structures in the lysosomes are thought to be early secondary lysosomes. This study shows that the liver cells have a remarkably effective defense mechanism against iron overload, which consists in the possibility of keeping a constant cell sap ferritin plateau by continuous transport of excessive iron into lysosomes.

9092 ISOLATED LESIONS OF THE PORTA HEPATIS IN NONPENETRATING TRAUMA OF THE ABDOMEN: ARTERIOGRAPHIC DATA. (Fre.) Malvy, P.; Le Neel, J. C.; Leborgne, J.; Bourdon, J.; Mitard, D. (Hotel-Dieu de Nantes, 44035 Nantes Cedex, France). *Chirurgie* 103(7):507-517; 1977.

Three patients with traumatic injuries to the porta hepatis due to auto accidents, all of whom were subjected to arteriographic study, are discussed. A 51-yr-old man entered the hospital with multiple trauma including fractures. He was conscious, and his blood pressure was normal at time of admission, but he went into severe shock 3 hr later. At emergency surgery, hemorrhagic peritonitis due to a dissection of the porta hepatis and a wound of the hepatic artery was found; the artery was repaired. At follow-up 1-yr selective opacification of the hepatic artery showed good vascularization of the liver. A 48-yr-old man who entered the hospital in shock underwent exploratory surgery. Hemorrhagic peritonitis was present. A ruptured spleen was removed. A tear in the hepatic artery was not repairable, and the wounded section of the artery was excised and the two sections linked. However, the liver failed to regain normal color after the procedure. Arteriography performed several days after surgery revealed a tear in the common hepatic artery that had not been observed at surgery. The patient succumbed to an infection before further surgery could be performed. A 37-yr-old man entered the hospital in shock, which improved after blood transfusion. On the 5th day of hospitalization, a progressive icterus appeared, and liver function studies showed a slight increase in amylase, SGOT, and SGPT activities. Arteriography showed no anomalies of the splenic or hepatic arteries; however, flow in the gastroduodenal artery was interrupted. Surgery was performed and bile was observed in the peritoneal fluid. A tear in the gallbladder was found as well as a healed wound of the gastroduodenal artery. A cholecystectomy was performed with a choledoschojejuna anastomosis. The patient recovered and left the hospital 2 months after the accident. Lesions of the porta hepatis are rare, particularly in the absence of associated hepatic damage.

9093 TOXICOLOGY OF COPPER. (Eng.) Hill, C. H. *In: Advances in Modern Toxicology: Toxicology of Trace Elements.* (New York: John Wiley & Sons): 2:123-127; 1977.

Copper toxicology in humans and animals is reviewed. As this metal is encountered in nature, its toxicity is relatively low except in two special circumstances: humans with Wilson's disease are especially sensitive to copper and sheep can be afflicted with copper toxicosis under practical husbandry practices. Ingested copper is absorbed from the stomach and upper small intestine, passes into the blood where it is probably bound to albumin, and passes to the liver. The liver concentrates the copper first in the cytosol, then in the mitochondria, and finally in the nuclei. It reappears in the plasma in the form of ceruloplasmin. The major excretory pathway is via the bile. In Wilson's disease, the liver, kidneys, brain, and cornea accumulate large amounts of copper. Domestic animals, particularly sheep, are more likely to sustain copper toxicosis than are humans, because of copper-containing salt licks and deliberate copper feeding. Sheep poisoning is characterized by the gradual hepatic accumulation of copper over several weeks or months with no outward symptoms, followed by hemolysis, hemoglobinemia, and hemoglobinuria accompanied by the development of jaundice; these occur coincident with the release of copper from the liver. Calves are less sensitive to copper poisoning and only develop symptoms after ingesting 10-fold more copper than required to poison sheep. Copper toxicosis in swine may be related to an induced deficiency of zinc and iron, since these elements have been shown to prevent the jaundice, elevated aspartate transaminase levels, and anemia seen in pigs fed 750 ppm copper. Rats are apparently insensitive to copper poisoning. It appears that copper toxicosis is not a major problem: in humans, the ingestion of excessive copper salts leads immediately to vomiting, whereas animals have a great capacity to store copper in the liver.

9094 CLINICAL DIAGNOSIS OF HEPATOCELLULAR CARCINOMA. (Eng.) Okuda, K.; Kubo, Y. (Chiba Univ. Sch. Medicine, Chiba, 280, Japan). *Kurume Med. J.* 24(Suppl.):S81-S112; 1977.

Clinical features significant for the diagnosis of hepatocellular carcinoma (HCC) and the value of alpha-fetoprotein (AFP) determination and of celiac angiography in diagnosing this disease are presented. Abdominal fullness and distention were early symptoms in 44% of 134 patients with HCC, and abdominal pain occurred in 41.8%. Other symptoms were similar to those in liver cirrhosis. In one patient, a firm mass was felt in the right upper quadrant beside a

normal liver edge; at autopsy, the lobe was found to be completely replaced by tumor. In other patients, localization of the tumor is difficult because of displacement of other organs, such as the spleen. Laboratory studies that are important in the diagnosis include alkaline phosphatase, SGOT, and SGPT levels; the SGOT/SGPT ratio; and LDH and AFP levels. The SGOT/SGPT ratio increases gradually during the advancement from chronic liver disease to cirrhosis and from cirrhosis to HCC; the ratio increases sharply during the weeks before death. The authors' own experience as well as the results of previous studies suggest that the AFP level is not always a reliable indicator of HCC. Liver sciniscan is regarded as one of the most reliable diagnostic tools in liver cancer. However, HCC arising in a cirrhotic liver is not readily detected by this method alone. The use of ^{99}Tc-labeled colloid is superior to that of ^{198}Au-colloid in this situation, and the right lateral, posterior, left lateral, and anterior positions should all be used. HCC can sometimes be differentiated from metastatic carcinoma because the parenchyma surrounding the tumor in HCC has low activity, and the defect is not very discrete. Celiac and hepatic angiography often disclose small lesions that have escaped detection by scanning; the former techniques also provide information on the gross form of the tumor. One of the most characteristic angiographic findings in HCC is the presence of arterio-portal shunts. The smallest lesion detected by angiography in the authors' experience was 4 x 4 cm. The authors suggest that three other types of HCC should be recognized (cirrhotic, cholestatic, and hepatitis) aside from the five previously presented in the literature (frank, occult, febrile, acute abdominal, and metastatic).

9095 CELL-MEDIATED CYTOTOXICITY AGAINST HUMAN LIVER LIPOPROTEIN IN LIVER DISEASE [Abstract]. (Eng.) Vogten, A. J. M.; Shorter, R. G.; Summerskill, W. H. J. (State Univ. Hosp., Utrecht, Netherlands). *Digestion* 16(4):319-320; 1977.

9096 CATABOLISM OF C1q AND C3 IN CHRONIC LIVER DISEASE [Abstract]. (Eng.) Potter, B. J.; Elias, E.; Thomas, H. C.; Sherlock, S. (Royal Free Hosp., Hampstead, London, England). *Digestion* 16(4):356; 1977.

9097 ACUTE FATTY LIVER OF PREGNANCY WITH SURVIVAL OF MOTHER AND CHILD: A CASE REPORT. (Eng.) Ylostalo, P.; Jouppila, P.; Rasanen, O.; Hynynen, T. (Dept. Obstetrics and Gynecology, Univ. Oulu, 90220 Oulu 22, Finland). *Eur. J. Obstet. Gynecol.* 7(6):351-356; 1977.

9098 HEPATIC TUMORS, SURGICAL TREATMENT AND ITS RESULTS. (Eng.) Lin, T. Y. (Natl. Taiwan Univ. Hosp., Taipei, Taiwan, Republic of China). *Jpn. J. Surg.* 7(4):189-198; 1977.

9099 RISKS OF CORTICOSTEROID THERAPY IN LIVER DISEASE. (Eng.) Tytgat, G. (No affiliation given). *Scand. J. Gastroenterol.* 12(Suppl. 47): 46-49; 1977.

9100 RISKS OF LIVER BIOPSY. (Eng.) Renger, F. (No affiliation given). *Scand. J. Gastroenterol.* 12(Suppl. 47):26-27; 1977.

9101 SPONTANEOUS RUPTURE OF A HEPATOMA. (Eng.) Singh, S.; Dhall, J. C.; Lal, M. M.; Sekhon, G. S. (Medical Coll. Hosp., 100 Doctors Hostel, Rohtak, Haryana, India). *Int. Surg.* 63(1): 45-46; 1978.

9102 FAMILIAL HBsAg-POSITIVE HEPATOMA: TREATMENT WITH ORTHOTOPIC LIVER TRANSPLANTATION AND SPECIFIC IMMUNOGLOBULIN. (Eng.) Johnson, P. J.; Wansbrough-Jones, M. H.; Portmann, B.; Eddleston, A. L. W. F.; Williams, R.; Maycock, W. D'A.; Calne, R. Y. (King's Coll. Hosp. and Medical Sch., London SE5, England). *Br. Med. J.* 1(6107): 216; 1978.

9103 HEPATIC DYSFUNCTION IN CONGESTIVE HEART FAILURE SIMULATING HEPATITIS. (Eng.) Field, R. S.; Meyer, G. W. (U.S. Air Force Medical Center, Keesler Air Force Base, MS 39534). *South. Med. J.* 71(2):221-222; 1978.

9104 URINARY TOTAL CATECHOLAMINES IN PREGNANT FEMALES WITH BILHARZIAL HEPATIC FIBROSIS. (Eng.) Abdel Aziz, M. T.; Abdel Kader, M. M.; Talaat, M.; Abdel Hamid, H.; El Fiki, A. F.; El Massry, A. A.; Gobba, S. A. (Faculty Medicine, Kasr El-Ainy St., UAR-Kairo, Egypt). *Dtsch. Gesundheitsw.* 33(4):185-186; 1978.

9105 "VASCULAR" BENIGN LIVER CYST IN CHILDREN: REPORT OF TWO CASES. (Eng.) Rosch, J.; Mayer, B. S.; Campbell, J. R.; Campbell, T. J. (Univ. Oregon Health Sciences Center, 3181 S.W. Sam Jackson Park Rd., Portland, OR 97201). *Radiology* 126(3):747-750; 1978.

9106 NEW CELLS FOR OLD. (Eng.) Anonymous. (No affiliation given). *Lancet* 1(8056): 133-134; 1978.

9107 HEPATIC GLYCOGEN SYNTHETASE DEFICIENCY: DEFINITION OF SYNDROME FROM METABOLIC AND ENZYME STUDIES ON A 9-YEAR-OLD GIRL. (Eng.) Aynsley-Green, A.; Williamson, D. H.; Gitzelmann, R. (John Radcliffe Hosp., Headington, Oxford OX3 9DU, England). *Arch. Dis. Child.* 52(7):573-579; 1977.

9108 TUBERCULOUS LIVER ABSCESS. (Eng.) Rab, S. M.; Zakaullah Beg, M. (Jinnah Postgraduate Medical Center, Karachi 35, Pakistan). *Br. J. Clin. Pract.* 31(10):157-158; 1977.

9109 CASE REPORT: PELIOSIS HEPATIS IN A CHILD. (Eng.) Bank, J. I.; Lykkebo, D.; Hagerstrand, I. (Odense Univ. Hosp., Odense, Denmark). *Acta Paediatr. Scand.* 67(1):105-107; 1978.

9110 ANGIOGRAPHY OF THE REGENERATING LIVER AFTER LOBAR RESECTION. (Eng.) Karp, W.; Tylen, U. (Univ. Hosp., S-221 85 Lund, Sweden). *Acta Radiol. [Diagn.] (Stock.)* 18(5):571-580; 1977.

9111 NECROTIC LIVER CELL ADENOMA--A RARE CAUSE OF THE ACUTE SURGICAL ABDOMEN. (Eng.) Fogler, R.; Weiner, E.; Lanter, B. (No affiliation given). *Abdom. Surg.* 20(1):11-13; 1978.

9112 MANAGEMENT OF INFERIOR VENA CAVAL INJURY IN LIVER TRAUMA. (Eng.) McMaster, P.; Tolley, M. (Addenbrooke's Hosp., Cambridge, England). *J. R. Coll. Surg. Engl.* 59(6):502-506; 1977.

9113 PREOPERATIVE AND POSTOPERATIVE CARE. (Eng.) Stone, H. H. (Emory Univ. Sch. Medicine, 69 Butler St., Atlanta, GA 30303). *Surg. Clin. North Am.* 57(2):409-419; 1977.

9114 GRAY SCALE ECHOGRAPHIC PATTERNS OF HEPATIC METASTATIC DISEASE. (Eng.) Scheible, W.; Gosink, B. B.; Leopold, G. R. (Univ. California Medical Center, 225 W. Dickinson St., San Diego, CA 92103). *Am. J. Roentgenol.* 129(6):983-987; 1977.

9115 CYSTS AND BENIGN TUMORS OF THE LIVER. (Eng.) Walt, A. J. (Wayne State Univ. Sch. Medicine, Detroit, MI). *Surg. Clin. North Am.* 57(2):449-464; 1977.

See also, 8713, 8781, 8783, 8784, 8786, 8791, 8792, 8793, 8794, 8809, 8820, 8926, 8972, 9042, 9129, 9131, 9132, 9134, 9141, 9152, 9154, 9161, 9171, 9172, 9176, 9195, 9217, 9224, 9245, 9246, 9248, 9251.

LIVER AND BILIARY TRACT

Hyperbilirubinemic States

9116 CHANGES OF SERUM GONADOTROPIN CONCENTRATIONS IN PREMATURE BABIES SUBMITTED TO PHOTOTHERAPY. (Eng.) Lemaitre, B.; Toubas, P. L.; Guillot, M.; Dreux, C.; Relier, J. P. (Hopital St.-Louis, rue de la Grange-aux-Belles, F-75010, Paris, France). *Biol. Neonate* 32(3/4):113-118; 1977.

To determine whether phototherapy affects plasma gonadotropin levels, follicle stimulating hormone (FSH) and luteinizing hormone (LH) levels were determined in eight premature infants who were treated for 2-8 days for hyperbilirubinemia and compared with those in six premature untreated infants without jaundice. In the four girls subjected to phototherapy, there were significant increases in plasma FSH ($p<0.001$) and LH ($p<0.01$) levels during the 3rd and 4th weeks after birth compared with levels in the 1st and 2nd weeks. FSH and LH levels also increased significantly during the 3rd and 4th weeks after birth in the treated newborn girls compared with the levels in controls (FSH $p<0.02$, LH $p<0.05$). In boys who underwent phototherapy, only the LH levels increased during the 3rd and 4th weeks. In both sexes, the rise in gonadotropin levels appeared between 8 and 10 days after the end of phototherapy. No significant changes in gonadotropin levels were observed in any of the controls. Constant phototherapy and/or the sudden stopping of the treatment in the jaundiced premature female newborn is responsible for a marked and transient elevation of gonadotropin.

Nonviral Hepatitis and Hepatic Injury

LIVER AND BILIARY TRACT

9117 METHOXYFLURANE HEPATITIS. (Eng.) Min, K. W.; Cain, G. D.; Sabel, J. S., Gyorkey, F. (Veterans Admin. Hosp., Houston, TX 77211). *South. Med. J.* 70(11):1363-1364; 1977.

A case of fulminant hepatitis due to repeated sniffing of methoxyflurane as a euphoriant is presented. The patient, a 42-yr-old man, presented with nausea, vomiting, icterus, pyrexia, lethargy, and vague, intermittent midabdominal pain. He had a history of heavy alcohol use but had consumed no alcohol for the last 3 yr. He admitted sniffing methoxyflurane for the last 2 months. The patient died in hepatic failure due to submassive hepatic necrosis 3 weeks after hospitalization. Fluorinated anesthetics should be considered as a causative agent in cases of fulminant hepatitis, especially in patients who have easy access to such drugs.

9118 POLYURIC RENAL FAILURE AND HEPATITIS ASSOCIATED WITH METHOXYFLURANE ANESTHESIA. (Eng.) Kalkay, N. M. (Brooklyn-Cumberland Medical Center, Brooklyn, NY). *N.Y. State J. Med.* 77(14):2265-2269; 1977.

A case is presented in which both of the toxic effects of methoxyflurane (hepato- and nephrotoxicity) occurred concurrently, and it is compared with similar cases from the literature. A 62-yr-old man underwent two episodes of retinal surgery 4 weeks apart in which methoxyflurane anesthesia was used. Renal injury manifested immediately: on the day of the second surgery, the patient developed a fever of 100.4 F that lasted 3 days, complained of weakness, dizziness, and excessive thirst, and was noted to have dehydration and orthostatic hypotension. Acute polyuric renal failure lasted 31 days. Hepatic injury began 12 days after the second surgery, manifested by low-grade fever, enlarged and tender liver, elevated transaminase level, and prolonged prothrombin time; it persisted 16 days. The hospital course was complicated by pericarditis, severe hypocalcemia, fluid and electrolytes imbalance, tachyarrhythmia, hypoalbuminemia, hypoprothrombinemia, and anemia. Peritoneal dialysis for azotemia and pericardial involvement was required intermittently for 2 weeks, and liver biopsy could not be done. Prior renal or hepatic disease, other toxic medications, blood transfusion, and shock were ruled out etiologically, and viral hepatitis appeared unlikely. Only four other proved cases of this type with sufficient data are known in the literature. Two different pathogenetic mechanisms appear to be at work. The nature of the renal disease seems to be predominantly tubular, perhaps induced by the direct toxic effects of fluoride. The injury is transient, dose-related, can be produced in experimental animals, and is not induced by halothane. The hepatic injury appears to be induced by an immunologic hypersensitivity reaction, has an unpredictable outcome with a very high mortality rate (perhaps 58%), cannot be produced in experimental animals, and is induced by halothane. Even the safest anesthetic agents are not free of severe side-effects.

9119 HEPATITIS DUE TO PERHEXILINE MALEATE: DEVELOPMENT OF CIRRHOSIS AFTER INTERRUPTION OF DRUG IN TWO CASES. (Fre.) Beaugrand, M.; Chousterman, M.; Callard, P.; Camilleri, J. P.; Petite, J. P.; Ferrier, J. P. (Hopital Jean-Verdier, avenue du 14-Juillet, F 93140 Bondy, France). *Gastroenterol. Clin. Biol.* 1(10):745-750; 1977.

Two cases are reported of liver damage in patients who were chronic users of the drug perhexiline maleate (Pexid). A 66-yr-old man had taken Pexid (200 mg/day for 23 months) to relieve pain associated with a myocardial infarction. He was hospitalized for vomiting and weight loss; his liver was hard and enlarged; and liver function studies and other laboratory tests showed abnormal liver function. Histological examination of the liver revealed sclerosis, inflammatory cell infiltration, and Mallory bodies in specimens of the centrilobular portion. Pexid was discontinued, and the patient was discharged from the hospital without signs of improvement in liver function tests. One month later he was rehospitalized with edema of the lower limbs, icterus, and hepatic encephalopathy, and he died within 2 weeks. A 53-yr-old man used Pexid (300 mg/day for 9 months) together with two other drugs for anginal pain. He was hospitalized for hepatomegaly and weight loss; liver function studies showed elevation of SGOT and SGPT levels and prolonged BSP retention. Mallory bodies and fatty degeneration were observed on microscopic examination of liver biopsies. Pexid was discontinued, and the patient was treated with prednisone. Liver function returned to normal, and histological examination revealed reduced inflammation, micronodular cirrhosis, but no Mallory bodies or hepatocytic lesions 6 months later. Both cases mimicked alcoholic hepatitis initially. The first patient had no history of alcohol consumption, whereas the second consumed no more than half a liter per day.

9120 TOXIC HEPATOSIS DUE TO SALICYLATES IN CHILDREN. (Ger.) Hinkel, G. K.; Korth, G.; Lorenz, K. (Kinderklinik, Medizinische Akademie "Carl Gustav Carus," Fetscherstr. 74, DDR-8019 Dresden, E. Germany). *Dtsch. Gesundheitswes.* 32(35): 1642-1644; 1977.

Three children treated with high doses of the salicylate Micristin (104-150 mg/kg/day) developed toxic hepatosis, which was also confirmed bioptically in one case. The clinical symptoms were mild, but the serum transaminase level was increased considerably in all cases. After normalization of the serum transaminase level, the Micristin therapy was continued at reduced dose (65 mg/kg/day) in one patient without any sign of intolerance. The findings indicate that toxic hepatosis due to high doses of salicylates occurs more frequently than has been assumed before and that the salicylate dose should be limited to 70 mg/kg/day. The serum transaminase level should be controlled regularly during salicylate therapy. Following the resolution of salicylate hepatitis, the salicylate therapy may be resumed after an interval but at a reduced dosage.

9121 CHRONIC HEPATIC INFLAMMATION AND FIBROSIS DUE TO LOW DOSES OF PARACETAMOL. (Eng.) Bonkowsky, H. L.; Mudge, G. H.; McMurtry, R. J. (Veterans Admin. Center, White River Junction, VT 05001). *Lancet* 1(8072):1016-1018; 1978.

The case of a patient in whom liver injury developed after chronic ingestion of paracetamol in therapeutic doses is presented. Chronic hepatic necrosis developed in a 53-yr-old man who had been taking 4 g of paracetamol daily for about 1 yr (cumulative dose 1,700 g). Liver biopsy done 23 days after the drug was stopped showed prominent diffuse central necrosis and portal changes. Repeat biopsy 5 months later showed chronic active hepatitis. This prompted anti-inflammatory treatment, with subsequent improvement in liver histology. Liver concentrations of reduced glutathione and paracetamol metabolism were assessed 1-1.5 yr after the drug was stopped and were normal; the basis for this patient's drug sensitivity is thus unclear. In some patients, chronic ingestion of therapeutic doses of paracetamol may produce hepatic necrosis and hepatitis that persist long after the drug has been discontinued.

9122 HALOTHANE HEPATITIS IN CHILDREN [Letter to Editor]. (Eng.) Crowe, G. R. (Struan House, Cnr Brimage St. and Wood Terrace, Whyalla, South Australia 5600, Australia). *Med. J. Aust.* 1(21):794; 1977.

9123 THE EFFECT OF ACETYLATION (AC) STATUS ON CHOLESTASIS AND HEPATOCELLULAR DAMAGE (HCD) IN ISONIAZID (INH) INDUCED LIVER DISEASE [Abstract]. (Eng.) Dickinson, D. S.; Hirschowitz, B. I.; Bailey, W. C.; Gibson, R.; Hodgkin, M. M. (Univ. Alabama Sch. Medicine, Birmingham, AL). *Gastroenterology* 72(5, Part 2):1049; 1977.

9124 FIRST CASES OF PYRROLIZIDINE ALKALOID POISONING IN THE UNITED STATES [Abstract]. (Eng.) Huxtable, R.; Stillman, A.; Ciaramitaro, D. (Univ. Arizona Health Science Center, Tucson, AZ 85724). *Pharmacologist* 19(2):181; 1977.

See also, 9184.

LIVER AND BILIARY TRACT

Acute Viral Hepatitis

9125 DIFFERENCES IN THE BIOCHEMICAL COURSE OF HBsAg-NEGATIVE AND HBsAg-POSITIVE HEPATITIS TITIS. (Ger.) Strohm, W. D.; Hossenfelder, E.; Stille, W. (Zentrum der Inneren Medizin, Universitat Theodor-Stern-Kai 7, D-6000 Frankfurt/Main 70, W. Germany). *Z. Gastroenterol.* 16(1):13-23; 1978.

A comparison of 73 hepatitis B surface antigen (HBsAg)-negative patients with 94 HBsAg-positive patients, all of whom had been free of other diseases that could influence the course of hepatitis prior to contracting the disease, was carried out to determine whether the course of the disease differed in these two groups. SGOT, SGPT, and bilirubin levels were higher in HBsAg-positive patients than in HBsAg-negative patients, and these levels remained higher for a longer time in the former group. During the acute stage, 56% of HBsAg-positive patients had an initial increase in transaminase activity, generally with an increase in the de Ritis quotient (SGOT:SGPT), and 15% of the patients reached a transaminase activity plateau; only 22% of the HBsAg-positive patients had a course similar to that of the HBsAg-negative patients. The median duration of the initial transaminase plateau in patients with hepatitis B was 17.9 ± 7.2 days; the duration of HBsAg-positive hepatitis was longer than that of HBsAg-negative hepatitis by more than 10 days. However, the duration of the disease in HBsAg-negative patients was appreciably longer in patients with transaminase recidivism with an increase in bilirubin. In HBsAg-negative patients, SGOT and SGPT values began to decrease immediately following the development of icterus. Laboratory determinations in the first days after the development of icterus were essentially the same in both types. The different biochemical behavior of HBsAg-negative and HBsAg-positive hepatitis may be due to differing immunological reactions of the liver.

9126 HISTOCHEMICAL DEMONSTRATION OF HBsAg IN LIVER BIOPSIES WITH ORCEIN STAINING.

(Ita.) Esposito, R.; Pollavini, G.; Randone, G.; De Lalla, F. (Istituto di Malattie Infettive dell'Universita di Milano, Milano, Italy). *Boll. Ist. Sieroter. Milan* 56(4):316-320; 1977.

The histochemical detection of hepatitis B surface antigen (HBsAg) was demonstrated using orcein stain. Liver biopsies were obtained from 30 HBsAg-seronegative and 33 HBsAg-seropositive individuals (5 were normal and 58 had liver diseases). The tissues were stained according to the modifications of Deodhar. In none of the HBsAg seronegative cases was positive staining obtained with orcein. Of the 33 HBsAg seropositive cases, 18 had hepatocytes whose cytoplasm was stained red-brown: 4/7 from patients with chronic persistent hepatitis, 5/9 from patients with chronic aggressive hepatitis, 6/11 from patients with chronic aggressive hepatitis with cirrhosis, and 3/6 from HBsAg-positive individuals without identifiable liver disease.

9127 SCANNING ELECTRON MICROSCOPY OF THE BILE DUCTULE. (Eng.) Itoshima, T.; Yoshino, K.; Yamamoto, K.; Ohta, W.; Kubota, M.; Ukida, M.; *et al.* (Okayama Univ. Medical Sch., Okayama, 700, Japan). *Gastroenterol. Jpn.* 12(6):476-482; 1977.

Scanning electron microscopy was used to study the bile ductule in a liver needle biopsy specimen from a 29-yr-old man in the convalescent stage of acute hepatitis, the onset of which had occurred 6 months earlier. The sinusoids, Disse spaces, portal triad vessels, tissue spaces, and bile ductule, which were all cleaned by puncture perfusion, were clearly demonstrated. The air crackings tended to occur between the cells and disclosed the intercellular surfaces. The ductule, portal triad, and portal vein branch were 13, 120, and 50 μm in outer diameter, resp. The ductule had sinusoids at the lobule side and tissue spaces and a 7-μm capillary at the portal side. The antiluminal border of the ductule was rather smooth and devoid of microvilli. The ductular lumen was surrounded by two ductular cells (each 18 μm in length), was 1.6-2.7 μm in diameter, and was ampullary dilated (2.7 μm in diameter) at the canalicular side. The canalicular-ductular junction was not evident. Intracytoplasmic diverticula with 0.9-μm diameter orifices were present in the lumen. Microvilli (0.4-0.6 μm long and 0.1 μm thick) were present on the luminal surface of the ductular cell (about 1,500/cell), being denser at the marginal flap and in the diverticula. A single cilium (0.15 μm wide at the base, 0.10 μm wide at the trunk, and 7-15 μm long) arose from a recess at the canalicular side of the ductular cell, and the free end was on the bile duct side. The distance from the luminal to the antiluminal side of the intercellular surface of the ductular cell was 5-6 μm. The outer area of the intercellular surface was undulated but without interdigitation. Occasionally, a cell with thin processes was intercalated between the ductular cells at the edge of the basal portion. The ductular cell surfaces corresponding to the intercalated cell were smoothly depressed and formed a canal 3 μm in diameter parallel to the long axis of the ductule.

9128 CHANGES IN AMINOPHENAZONE METABOLISM IN CHILDREN WITH ACUTE HEPATITIS. (Ger.) Windorfer, A., Jr.; Muller, G.; Stehr, C. (Kinderklinik und Poliklinik, Technische Universitat, Kolner Platz 1, 8000 Munich 40, W. Germany). *Dtsch. Med. Wochenschr.* 102(41):1460-1464; 1977.

The serum concentrations of aminophenazone (AP) were measured in 23 3- to 13-yr-old children with acute viral hepatitis and in 23 age-matched controls with normal livers following the administration of single doses of AP (8-22 mg/kg p.o.), as well as in 5 healthy young adults who received 8-17 mg/kg p.o. of AP. The control children received AP for acute attacks of fever. The serum half-life of AP was 2.85 ± 0.89 hr in the adult controls, 2.79 ± 0.53 hr in the control children, and 6.71 ± 3.34 hr in the children with hepatitis; the difference between the patients and controls was statistically significant ($p<0.1$). The serum AP concentration was highest 2 hr after administration, being somewhat higher in the control children than in the patients with hepatitis, but the difference was not significant due to the wide scatter of the data. The serum half-life of AP increased with increasing SGPT level ($r=0.9$) in the patients, but there was no correlation between the half-life and the dose in either control group. The maximum serum concentration of AP correlated with the dose in the control children ($r=0.84$), but only a weak correlation was seen in the patients ($r=0.38$). The findings indicate the interference of hepatopathy with AP metabolism.

9129 CLINICAL SIGNIFICANCE OF e-ANTIGEN/ANTI-e, WITH SPECIAL REFERENCE TO HBc-ANTIGEN IN THE LIVER. (Eng.) Furuta, S.; Kiyosawa, K.; Nagata, A.; Koike, Y.; Sahara, T.; Furukawa, K.; *et al.* (Dept. Internal Medicine, Shinshu Univ., Matsumoto, 3-1-1, Nagano-prefecture, 390 Japan). *Gastroenterol. Jpn.* 12(6):460-465; 1977.

The prevalence of hepatitis B e antigen (HBeAg), antibody to HBeAg (anti-HBe), and HB core antigen (HBcAg) was evaluated in 34 asymptomatic HB surface Ag (HBsAg) carriers, 8 patients with acute hepatitis, 16 with chronic persistent hepatitis, 36 with chronic active hepatitis, 35 with cirrhosis, and 23 with hepatoma, all of whom were HBsAg-positive. The frequency of HBeAg in these patient groups was 38.2, 25.0, 12.5, 33.3, 5.7, and 0%, resp., and of anti-HBe was 23.6, 50.0, 56.2, 61.1, 80.0, and 78.3%, resp. Among 22 asymptomatic HBsAg carriers, HBcAg was detected in the liver of 4/8 with HBeAg in the serum, 2/9 with anti-HBe in the serum, and 2/5 with neither HBeAg nor anti-HBe in the serum. Among 48 patients with chronic liver diseases, HBcAg was detected in the liver of 4/5 with HBeAg in the serum, 1/5 with anti-HBe in the serum, and 23/38 with neither HBeAg nor anti-HBe in the serum. The serum titer of antibody

to HBcAg (anti-HBc) was lower in sera positive for anti-HBe than in sera positive for HBeAg ($p<0.05$), indicating that there may be a decreased level of HB virus replication in patients with anti-HBe. The results emphasize that the investigation of HBeAg and anti-HBe is mandatory for the evaluation of the prognosis of asymptomatic HBsAg carriers and of patients with chronic hepatitis.

9130 BEHAVIOR AND CLINICAL SIGNIFICANCE OF THE eAg/anti-e SYSTEM IN HBsAg CARRIERS. (Ita.) Pastore, G.; Dentico, P.; Fera, G.; Angarano, G.; Schiraldi, O. (Istituto di Malattie Infettive dell'Universita di Bari, Bari, Italy). *Boll. Ist. Sieroter. Milan.* 56(4):333-338; 1977.

The relationship between hepatitis B e antigen (HBeAg), antibody to HBeAg (anti-HBe), and various forms of liver pathology caused by HB virus (HBV) was studied. Radioimmunoassay and radial immunodiffusion were used to determine HB surface Ag (HBsAg), antibody to HBsAg (anti-HBs), HBeAg, and anti-HBe levels in sera from 315 patients (154 with acute hepatitis, 44 HBsAg-positive asymptomatic subjects, and 117 with chronic hepatitis) drawn weekly during the period of recovery and at less frequent intervals during outpatient follow-up. For those with acute hepatitis, 31/82 who were cured had HBeAg in their sera during the acute phase, 8/82 had anti-HBe in their sera at this time, and all were positive for HBsAg. Six months later, none were positive for HBsAg, 50 had anti-HBs, 2 had HBeAg in their sera, and 25 had anti-HBe antibodies. Of the patients with protracted cases of acute hepatitis, 20/20 had HBsAg, 13/20 had HBeAg, and none had anti-HBe or anti-HBs in their sera during the acute phase. Six months later, 12/20 still had HBsAg, 2/20 had anti-HBs, 8/20 had HBeAg, and none had anti-HBe antibodies. Of the 52 HBsAg-negative patients with acute hepatitis, none were positive for HBeAg or anti-HBe. Of the 117 patients with chronic hepatitis, 28 had neither HBsAg, anti-HBs, HBeAg, nor anti-HBe. Of the 30/117 with chronic persistent hepatitis, 19/30 were HBsAg-positive (10/19 also had HBeAg, and 6/19 also had anti-HBe); 4/30 were anti-HBs-positive (2/4 also had HBeAg, and 1/4 also had anti-HBe). In the 30/117 with chronic aggressive hepatitis type A, 20/30 were HBsAg-positive (17/20 also had HBeAg, and 2/20 also had anti-HBe); 6/30 were anti-HBs-positive (3/6 also had HBeAg, and none had anti-HBe). Of the 9/117 with chronic aggressive hepatitis type B, 7/9 were HBsAg-positive (all also had HBeAg, and none had anti-HBe); 1/9 had anti-HBs and HBeAg but not anti-HBe. Of the 48/117 with cirrhosis, 22/48 were HBsAg-positive (9/22 also had HBeAg, and 1/22 also had anti-HBe); 10/48 were anti-HBs-positive (2/10 also had HBeAg but none had anti-HBe). Among the 44 HBsAg-positive but asymptomatic patients, only one, who was diagnosed as having chronic aggressive hepatitis, was HBeAg-positive, but 24 (17 with a history of acute hepatitis) had anti-HBe. The results confirm ån association between HBV infection and the presence of HBeAg and anti-HBe in the serum.

9131 PRESENCE OF SERUM CREATINE KINASE ISOENZYMES IN LIVER FAILURE WITH CEREBRAL INVOLVEMENT. (Eng.) Lemberg, A.; Werber, D. H.; Gilardoni, A.; Magrino, H. J. (Sch. Pharmacy and Biochemistry, Buenos Aires Univ., Buenos Aires, Argentina). *Clin. Chim. Acta* 80(2):385-390; 1977.

The presence of creatine kinase of the type that is derived from brain cells (BB-CK) in patients with severe encephalopathy and coma is reported. Twelve patients with clinical and biochemical evidence of hepatic failure, one diabetic with hyperglycemia and acidosis, and one with meningococcus purulent meningitis were studied. BB-CK was detected in the sera of all four patients with liver coma due to acute viral hepatitis. BB-CK occurred in three of four patients with cirrhosis and coma but in neither of two patients with chronic active hepatitis and coma. BB-CK was not detected in the sera of any of the remaining patients, all of whom also had severe encephalopathy. Only one patient with acute hepatitis survived, and in this patient the BB-CK disappeared. The enzyme was absent before the onset of encephalopathy in one patient with cirrhosis, but appeared when her condition deteriorated to grade IV coma. The concentrations of the enzyme ranged from 45 to 306 U/l in the patients with acute hepatitis and from 11 to 71 U/l in the patients with chronic liver disease. It seems clear that patients in this study can be divided into three groups: those with acute viral hepatitis, who show high BB-CK content in the serum; those with cirrhosis and chronic active hepatitis, who had either low serum BB-CK levels or none; and those with secondary liver involvement or with no liver involvement but with a severe general condition, in whom no BB-CK is found. Measuring CK isoenzymes in patients with severe liver pathology may help to measure the severity of the disease state.

9132 MODIFICATIONS OF FACTOR VIII RELATED ANTIGEN IN HEMOPHILIACS WITH ACUTE HEPATITIS AND SUB-CLINICAL LIVER DISEASE. (Eng.) Sultan, Y.; Maisonneuve, P.; Simeon, J.; Intrator, L. (Hopital Saint-Louis 2, Place du Dr. A. Fournier 75475, Paris Cedex 10, France). *Scand. J. Haematol.* (Suppl. 30):16-26; 1977.

Modifications in factor VIII-related antigen (FFRA) were investigated in three patients with hemophilia during the course of post-transfusion hepatitis. The patients were among 36 multitransfused hemophiliacs in whom the mean FFRA level was 148.5% of normal. The level in the 10 hemophiliacs with a normal SGPT level was 112.4% of normal, while that in the 26 hemophiliacs with elevated SGPT levels was 163.2% of the normal level. In the first patient, the FFRA level was 300% of normal 4 days after the onset of jaundice and reached 620% on the 18th day. At that time the bilirubin level was 14.0 mg/100 ml, and the SGPT level was 165 U. The ristocetin cofactor was 280% (less than half the FFRA level). By the 32nd day, the SGPT and

bilirubin levels were normal, and the FFRA level was near the normal range, although hepatitis B surface antigenemia persisted. Two-dimensional immunoelectrophoresis of the plasma on the 18th day showed abnormal results. FFRA appeared to be composed of two populations of FFRA molecules with different electrophoretic mobilities; this finding was confirmed by filtration of the cryoprecipitate on Sepharose 4B. The ability of the eluted protein to induce ristocetin aggregation in a washed platelet system was significantly reduced compared with the concentration of antigenic material. The FFRA level at the onset of disease in the second patient was 240% without modification of the procoagulant activity. The clinical symptoms rapidly resolved, and the liver function tests returned to normal. The FFRA gradually normalized. Immunodiffusion of FFRA during the acute phase showed a normal pattern. The third patient had a prolonged course of anicteric hepatitis. The FFRA was 180% during the second month, and this reached 210% after 8 months, during which time the transaminase level was consistently high. The ristocetin cofactor never increased during the course of the disease. Column chromatography of the patient's cryoprecipitate on Sepharose 4B showed the same abnormal elution pattern for FFRA and ristocetin cofactor seen in the first patient. These studies show that FFRA levels parallel the course of acute hepatitis and SGPT levels.

9133 RAPID TURNOVER SERUM PROTEINS IN FULMINANT HEPATITIS. (Eng.) Kobayashi, K.; Kameda, S.; Sugimoto, T.; Suzuki, K.; Nishimura, K.; Kato, Y.; *et al.* (Kanazawa Univ. Hosp., 13-1, Takaramachi, Kanazawa, Ishikawa, 920 Japan). *Gastroenterol. Jpn.* 12(6):455-459; 1977.

To find reliable indices for the early diagnosis of fatal cases of viral hepatitis, the prealbumin and α_2-heat stable(HS)-glycoprotein levels were measured by immunodiffusion and the Normotest was carried out using sera from 78 patients with biopsy- or necropsy-verified acute viral hepatitis. The patients were divided into three groups according to clinical and histological features: group A, 23 cases of subacute fulminant hepatitis with submassive necrosis and serious signs (e.g., mental disturbance, edema, ascites); group B, 15 cases with zonal or submassive necrosis without serious signs; and group C, 40 cases with spotty necrosis. Fourteen group A patient died. All tests were done before the appearance of hepatic coma or gastrointestinal bleeding. No significant differences were found in the prealbumin (half-life 1-2 days) level between fatal and surviving patients. The α_2-HS-glycoprotein (half-life 4-6 days) level was significantly lower in the fatal group A cases (21.9 ± 7.4 mg/dl) than in the surviving group A cases (37.4 ± 6.9 mg/dl, $p<0.05$); moreover, there was a significant ($p<0.05$) difference between group A overall (27.5 ± 7.4 mg/dl) and group B (60.9 ± 13.9 mg/dl) and an increased value among 30% of group C with respect to the α_2-HS-glycoprotein level. The Normotest showed significantly ($p<0.05$) lower values among fatal group A cases (10.7 ± 4.3%) compared with surviving group A cases (45.3 ± 19.6%) and among group A cases overall compared with group B cases ($p<0.05$). There was a significant correlation between the levels of α_2-HS-glycoprotein and the values of the Normotest both for the 44 cases of acute hepatitis ($\gamma=0.766$, $p<0.01$) and for the 15 cases of subacute fulminant hepatitis alone ($\gamma=0.852$, $p<0.01$). These results indicate that the values of α_2-HS-glycoprotein and the Normotest may be used for early identification of patients at high risk of death from viral hepatitis in whom aggressive therapy may be of value if instituted early enough.

9134 FOUR AUTOPSY CASES OF NEONATAL GIANT CELL HEPATITIS DIED SUDDENLY AND UNEXPECTEDLY FROM INTRACRANIAL HEMORRHAGE. (Eng.) Sugai, M.; Kono, R.; Hiroki, T.; Kunita, Y.; Tokudome, S. (Tokyo Medical Examiner Office, Tokyo, Japan). *Acta Pathol. Jpn.* 28(1):185-191; 1978.

The cases of four patients with neonatal giant cell hepatitis who died unexpectedly from intracranial hemorrhage are presented. The patients were one boy and three girls, aged 37 to 50 days old. The postnatal growth rate was retarded in all four infants. At autopsy, the most prominent features of the liver were turbidity and an icteric color on the cut surface. Histologically, the lobular structure was irregular, and the normal appearance of parenchymal cell cords was lost. Multinucleate cells were present in the center of the lobules. Bile canaliculi were absent or poorly preserved within these cells, and bile stasis was evident. There was massive iron pigment deposition in hepatocytes and Kupffer cells in the periportal area. These changes were present in all cases, but their severity differed between cases. The weight of the brain was increased because of the presence of edema in all patients. In most cases, the hemorrhage occurred subdurally due to a massive intracerebral hematoma in the region of the centrum semiovale.

9135 CHANGES OF THE ERYTHROCYTE MEMBRANE AND SERUM LIPIDS IN ACUTE HEPATITIS AND LIVER CIRRHOSIS. (Ger.) Pristautz, H.; Stubchen-Kirchner, H.; Fereberger, W. (Medizinische Universitatsklinik, Auenbruggerplatz 15, A-8036 Graz, Austria). *Wien. Med. Wochenschr.* 128(4):99-103; 1978.

Changes in erythrocyte membrane lipids and their possible relationship to serum lipids were studied in 33 patients with acute viral hepatitis, 11 patients with nondecompensated liver cirrhosis, and 11 healthy probands. The erythrocyte osmotic resistance (EOR) was normal in 10/33 patients with acute viral hepatitis and elevated in 23. The SGOT level was significantly increased in the hepatitis patients with elevated EOR compared with those with normal EOR (306 ± 45 mU/ml versus 147 ± 29 mU/ml). The SGPT, γ-glutamyltransferase, and alkaline phosphatase (AP) levels showed no correlation with elevated EOR. In the hepatitis pa-

tients with elevated EOR, the serum triglyceride level was noticeably but not significantly higher than that in the patients with normal EOR (210 ± 19 versus 158 ± 19 mg/100 ml), but the erythrocyte membrane cholesterol level was increased in 20/33 patients with elevated EOR and in only 1/33 patients with normal EOR. Among the cirrhotic patients, all four with elevated EOR showed increased erythrocyte membrane cholesterol levels, but there was no significant correlation between the serum enzyme levels and EOR. The lipid electrophoresis pattern was pathological in almost all hepatitis and cirrhosis patients with elevated EOR. The erythrocyte changes seen in viral hepatitis were already demonstrable a few days after onset of the symptoms, and always disappeared after the normalization of the transaminase levels. The findings indicate that the increased incorporation of cholesterol into erythrocytes in acute hepatitis is due to a disorder in hepatogenic apolipoprotein synthesis, rather than to excessive serum lipoprotein supply.

9136 ACUTE VIRAL HEPATITIS TYPE B IN CROATIA (YUGOSLAVIA). (Eng.) Fiser-Herman, M.; Mihaljevic, I.; Borcic, B. (Blood Transfusion Center of Croatia, Zagreb, Yugoslavia). *Acta Med. Iugosl.* 32(1):33-43; 1978.

9137 PREVALENCE OF AUSTRALIA ANTIGEN AND ANTIBODY AND ITS SUBTYPES IN INDIA. (Eng.) Dutta, R. N.; Dutta, A. K. (Armed Forces Transfusion Center, Dehli, India). *J. Indian Med. Assoc.* 69(8): 165-170; 1977.

9138 ACUTE VIRAL HEPATITIS. (Eng.) Woolf, I.; Williams, R. (King's Coll. Hosp., London, England). *J. Appl. Med.* 3(8):813-818; 1977.

9139 COMPLEMENT STUDIES IN SEVERE VIRAL HEPATITIS OF CHILDHOOD. (Eng.) Dupuy, C.; Dupuy, J. M. (INSERM U-56, Hopital d'Enfants, 78, rue General-Leclerc, 94270 Bicetre, France). *Pathol. Biol. (Paris)* 25(8):553-558; 1977.

9140 SERUM AND URINARY URIC ACID IN INFECTIOUS HEPATITIS. (Eng.) Raz, R.; Krivoy, N.; Rochman, J.; Eidelman, S. (Rambam Univ. Hosp., Haifa, Israel). *Isr. J. Med. Sci.* 13(12):1219-1221; 1977.

9141 THE RELATIONSHIP BETWEEN HEPATITIS B VIRUS INFECTION AND HEPATIC CELL CARCINOMA IN MOZAMBIQUE. (Eng.) Reys, L. L.; Purcell, R. H.; Holland, P. V.; Alter, H. J. (Faculty Medicine, Univ. Eduardo Mondlane, Mozambique). *Trop. Geogr. Med.* 29(3):251-256; 1977.

9142 ACQUIRED ENZYMOPATHY OF ERYTHROCYTE GLUCOSE-6-PHOSPHATE DEHYDROGENASE IN ACUTE VIRAL HEPATITIS. (Eng.) Kwiatkowska, J.; Kwiatkowska, D.; Andrzejak, R.; Zatonski, W. (Medical Sch., 10 Chalubinskiego, 50-368 Wroclaw, Poland). *Clin. Chim. Acta* 83(1/2):75-79; 1978.

9143 EARLY DIAGNOSIS OF HEPATIC INSUFFICIENCY IN PATIENTS WITH VIRUS HEPATITIS. (Rus.) Mikhailova, A. M.; Mel'nik, G. V. (Chernovtsy Medical Inst., Chernovtsy, USSR). *Vrach. Delo* (9): 137-140; 1977.

9144 RICKETTSIAL GRANULOMATOUS HEPATITIS: THE VALUE OF LIVER BIOPSY. (Fre.) Faure, J.; Escande, J. C.; Grataloup, G.; Massot, Ch.; Faure, H. (C.H.U. de Grenoble, F 38700 La Tronche, France). *Nouv. Presse Med.* 6(32):2896; 1977.

9145 CLINICO-IMMUNOLOGICAL CHARACTERISTICS OF VIRAL HEPATITIS WITH SPLENOMEGALY. (Rus.) Lenartovich, L. S. (Kiev Scientific Res. Inst. Infectious Diseases, Kiev, USSR). *Vrach. Delo* (11): 131-134; 1977.

See also, 9086, 9102, 9103, 9146.

LIVER AND BILIARY TRACT

Chronic Hepatitis

9146 STUDIES OF LYMPHOCYTOTOXICITY IN ACUTE AND CHRONIC LIVER DISEASE. (Eng.) Warnatz, H.; Gutmann, W.; Rosch, W.; Hommel, G. (Institut und Poliklinik f. klinische Immunologie der Universitat Erlangen-Nurnberg, Postfach 35 60, D-8520, Erlangen, W. Germany). *Z. Immunitaetsforsch.* 153(5):435-449; 1977.

The cytotoxicity of lymphocytes from 14 patients with chronic active hepatitis, 8 with chronic persistent hepatitis, 11 with acute hepatitis B, 12 with rheumatoid arthritis, and 32 normal controls was studied by a microcytotoxicity assay using ^{125}I-iododeoxyuridine-labeled embryonal liver cells and Chang cells as target cells. Unfractionated lymphocytes of the peripheral blood from patients with chronic active hepatitis and rheumatoid arthritis showed a high frequency of cytotoxic activity; the lymphocytotoxicity

in chronic active hepatitis was significantly increased in comparison with that in normal controls ($p<0.025$-0.05) at the effector cell to target cell ratios of 10:1 and 100:1. Specificity of the cytotoxic reaction to target cells could not be demonstrated. Addition of autologous serum to the cytotoxic assay blocked the lymphocytotoxicity in patients with chronic active hepatitis. A weak potentiating effect on lymphocytotoxicity was observed in patients with hepatitis B (HB) after addition of autologous serum. This reaction may be due to the presence of HB surface antigen (HBsAg) in the serum, since addition of HBsAg from other sources also increased the lymphocytotoxicity in HB patients ($p<0.05$). This effect was not observed either in HBsAg-positive or in HBsAg-negative patients with chronic active hepatitis or chronic persistent hepatitis.

9147 LIVER SPECIFIC AUTOANTIBODIES AND IMMUNOCOMPLEXES IN CHRONIC ACTIVE HEPATITIS. (Ita.) Realdi, G.; Trevisan, A.; Losi, C.; Tremolada, F.; Roul, A. (Istituto di Medicina Clinica, Policlinico dell'Universita, 35100 Padova, Italy). *Boll. Ist. Sieroter. Milan.* 56(5):476-488; 1977.

A direct immunofluorescent technique was used to detect IgG, IgM, complement component C_3, and hepatitis B surface antigen (HBsAg) on the membrane of isolated hepatocytes obtained from biopsies in three groups of selected patients: 12 patients with inflammatory liver diseases (chronic persistent hepatitis, chronic active hepatitis, alcoholic hepatitis, or cirrhosis), 7 patients with chronic immune complex diseases without liver damage (systemic lupus erythematosus, rheumatoid arthritis, Sjogren's syndrome, or idiopathic cryoglobulinemia), and 6 control patients with nonhepatic diseases. Autoantibodies directed against a liver-specific protein (LSP) were identified by an indirect immunofluorescent technique using isolated rabbit hepatocytes. HBsAg was detected on the hepatocyte surface only in two cases of chronic persistent hepatitis, where surface IgG and IgM were absent. In all other patients with inflammatory liver diseases and in all patients with immune complex diseases, membrane IgG and IgM were found. Only in the patients with chronic active hepatitis (CAH) was C_3 found on the liver cell membrane. The pattern of fluorescence was linear-granular (at different levels of focus) in HBsAg-negative CAH and in alcoholic hepatitis, and granular in all the other cases. Anti-LSP antibodies were detected in the sera of two patients with serological features of autoimmune CAH. These results confirm a possible pathogenic role of liver-specific autoimmunity in some cases of CAH but also point out the presence of immune complexes in the livers of all patients with circulating immune complexes, whether or not these patients have liver damage. In all these diseases, a clearance function of hepatocytes for circulating immune complexes may be postulated. The presence of complement on the hepatocyte surface in chronic active liver disease suggests a possible pathogenic role of some immune complexes, probably those formed by LSP and its antibody.

9148 CLINICAL SIGNIFICANCE OF THE VARIATIONS OF THE PLASMA PROTEINS IN CHRONIC HEPATITIS. (Ita.) Pugliese, V.; Picciotto, A.; Testa, R.; Mansi, C. (Istituto Scientifico di Medicina Interna, Universita di Genova, Genova, Italy). *Minerva Dietol. Gastroenterol.* 23(4):301-306; 1977.

Changes in serum proteins in chronic hepatitis are reviewed, and data on 193 patients with laparotomy- and biopsy-confirmed cases are presented. Transaminase, alkaline phosphatase, gammaglobulin, and prothrombin levels were evaluated in 45 patients with chronic persistent hepatitis (CPH) and in 148 with chronic aggressive (or evolutive) hepatitis (CAH). Transferin and α_1-antitrypsin levels were normal in 25/25 patients; the orosomucoide level was diminished in 12% of the cases; the α_2-macroglobulin level was increased in 56%; and the C'3 complement fraction was increased in 36%. The IgG, IgA, and IgM levels were increased in 36%, 40%, and 20% of the subjects, resp. The hepato-Quick test was altered in 65% of the patients. Serious deviations in fibrinoginemia were not observed. Fifty percent of the CAH patients had hypergammaglobulinemia, 45% had altered SGOT levels, 38% had altered SGPT levels, and 25% had altered alkaline phosphatase levels. The values for CPH patients were 25%, 30%, 25%, and 10%, resp. It is concluded that seroproteins alone are not a precise diagnostic or prognostic pool but are more useful when correlated with other clinical and morphological data.

9149 DYNAMICS OF STOMACH FUNCTION INDICES DURING GLUCOCORTICOID THERAPY FOR CHRONIC HEPATITIS AND LIVER CIRRHOSIS IN CHILDREN. (Rus.) Deputatova, L. D. (Dept. Pediatrics, S. M. Kirov Gorkii Medical Inst., Gorkii, USSR). *Vopr. Okhr. Materin. Det.* 21(9):36-41; 1976.

9150 EFFECT OF GLUCOCORTICOIDS ON THE CLINICAL COURSE AND SOME INDICES OF NITROGEN METABOLISM IN PATIENTS WITH CHRONIC VIRAL HEPATITIS. (Rus.) Melk, M. V. (Leningrad Inst. Pediatric Medicine, Leningrad, USSR). *Vrach. Delo* 6:137-139; 1976.

See also, 8828, 9079, 9080, 9085, 9086, 9096, 9126, 9129, 9130, 9131, 9137.

9151 SERUM ALPHA-FETOPROTEIN IN ALCOHOLIC LIVER DISEASE. (Fre.) Dedieu, P.; Simon, J.; Chatal, J. F.; Buzelin, F.; Le Portz, J.; Miniconi, P. (Hopital Saint-Jacques, C.H.R., F44035 Nantes Cedex, France). *Gastroenterol. Clin. Biol.* 2(1): 7-14; 1978.

To determine the frequency and importance of the elevation of serum alpha-fetoprotein (AFP) level and to correlate these levels with clinical, biological, and anatomical-pathological findings, the serum AFP level was measured by radioimmunoassay in 210 patients with alcoholic liver diseases and in 139 controls. The serum AFP level was significantly elevated ($p<0.001$) in all three patient groups compared with the level in the controls (6.41 ± 1.40 ng/ml); it was >10 ng/ml in 14% of the 41 patients with alcoholic hepatopathy without cirrhosis (average, 9.2 ± 8.5 ng/ml), in 20% of the 146 patients with alcoholic cirrhosis, and in 15/23 patients with alcoholic cirrhosis and primary liver carcinoma. Serum AFP levels higher than 100 ng/ml were observed only in the patients with cirrhosis and the 21 with hepatomas. Among the patients with alcoholic cirrhosis, the serum AFP level was significantly elevated in all subgroups (compensated cirrhosis, isolated icterus, and ascites plus edema, $p<0.001$, and ascites plus edema plus icterus, $p<0.01$) except in the 29 patients with hepatic encephalopathy. There was no significant correlation between serum AFP levels and clinical manifestations, biochemical disorders, or histological lesions in any patients. In abstinent patients, serum AFP, after a transient elevation, tended to decrease progressively and return to normal level in 3 weeks. No correlation was found between serum AFP level and the survival time of the 21 patients with hepatoma. In the two patients with colangioma, serum AFP level was normal. It is concluded that, in patients with alcoholic cirrhosis, a serum AFP level >100 ng/ml or one that fails to decrease after abstinence strongly suggests the development of hepatoma.

9152 SERUM GAMMA-GLUTAMYL TRANSPEPTIDASE ACTIVITY IN HEPATOBILIARY DISEASE: ROLE OF CHRONIC ALCOHOLISM. (Fre.) Faivre, J.; Lamblot, J.; Michalet, J. P.; Massart, J. P.; Klepping, C. (Hopital General, 1, rue de l'Hopital, F 21033 Dijon Cedex, France). *Lyon Med.* 239(2):55-59; 1978.

Serum gamma-glutamyltranspeptidase (γGT) activity was studied in 30 normal subjects, 30 nonalcoholic patients without hepatobiliary disease, 79 patients with alcoholic liver cirrhosis (36 confirmed by biopsy), 34 chronic alcoholics without liver pathology, 49 patients with liver cancer, 30 patients with biliary duct obstruction, and 19 patients with acute benign viral hepatitis to determine its diagnostic value in liver disease. Levels of serum alkaline phosphatase, prothrombin, total bilirubin, and SGPT were also measured. Patients who had liver cancer and who were also chronic alcoholics had the highest γGT levels. All patients who were chronic alcoholics had elevated serum γGT levels; abstinence from alcohol resulted in a drop to normal levels, but if cirrhosis was present, return to normal levels was not consistently observed. In patients who were not chronic alcoholics, there was a marked correlation between levels of alkaline phosphatase and γGT; the correlation was not observed in alcoholics. Elevated γGT levels may indicate liver cancer in nonalcoholic patients, but the effect of alcohol consumption on γGT levels obscures the diagnostic value of γGT levels in alcoholics. Measurement of γGT should not be substituted for that of SGPT levels in patients with viral hepatitis since the enzyme is not as specific an indicator for these patients.

9153 THE IMMUNOPATHOLOGY OF ALCOHOLIC HEPATITIS: RECENT FINDINGS. (Ita.) Lovisetto, P.; Actis, G. C.; Marchi, L.; Ferraris, R.; Biarese, V. (Istituto di Medicina Interna, Universita degli Studi Torino, Torino, Italy). *Minerva Med.* 68(62): 4147-4151; 1977.

Recent findings on the immunopathology of alcoholic hepatitis are reviewed and discussed. Epidemiological studies show that 30% of heavy drinkers have liver problems associated with alcohol, and 10% develop liver cirrhosis in 1-12 yr. The hepatopathy of the alcoholic cannot be attributed exclusively to the toxic effects of ethanol; immunologic factors are believed to play a leading role. Hypergammaglobulinemia (IgG, IgA, and IgM) has been noted in patients with liver cirrhosis. Serum IgA from these patients binds to Mallory bodies, and in the initial phase of alcoholic hepatitis, it is possible to find serum antibodies to Mallory bodies. In patients with hepatopathy from alcohol, antibodies to albumin have also been found. It is hypothesized that the specific antigenicity of albumin in these cases has been altered by acetaldehyde, a product of ethanol metabolism. As many as 50% of these patients have been reported to have a reduced skin reactivity to dinitrochlorobenzene, and many have a diminished blast transformation of lymphocytes in response to phytohemagglutinin. A correlation has been found between the inability of a patient's lymphocytes to form spontaneous rosettes with sheep RBC and the degree of alcoholic liver damage in cases of cirrhosis. The leukocyte migration inhibition test shows that WBC from these patients produce migration inhibition factor in response to Mallory bodies or homogenates of healthy livers. It has been demonstrated that the supernatant fraction from a culture of patient lymphocytes incubated with Mallory bodies augments the incorporation of proline into collagen. The therapeutic implications are to treat the patient with corticosteroids that act against the immune system and with colchicine, an inhibitor of collagen formation.

9154 ENTERAL LIPID ABSORPTION IN PATIENTS WITH PORPHYRIA CUTANEA TARDA AND IN ALCOHOLICS. (Ger.) Siklosi, C. (Universitats-Hautklinik Szeged, Szeged, Hungary). *Z. Hautkr.* 51(10):387-392; 1976.

Because lipid metabolism reacts promptly to any deterioration of hepatic and pancreatic function, enteral lipid absorption was investigated in 28 porphyria cutanea tarda (PCT) patients, in 21 chronic alcoholics, and in 20 dermatology patients without metabolic diseases (controls). Blood samples taken before as well as 2, 4, and 6 hr after the p.o. administration of Lipiodol Ultrafluid (an iodinated marker medium, 0.04 ml/kg body weight) were analyzed using potentiometry. Serum iodine levels decreased in the 6 hr following Lipiodol administration: by 82.14% in the PCT patients, 52.38% in the alcoholics, and 25% in the control group. The abnormally low value in the control group was attributed to the failure of some individuals to eat following Lipiodol intake. Thus, the Lipiodol test can provide information about the functional state of the small intestine mucosa only in cases of undisturbed digestive gland function. The advantages of the method include its simplicity, its very small probability of false positives, and it can provide information about the type of intermediary metabolism. However, this method provides reliable information only about the state of enteral absorption.

9155 COAGULATION FACTORS AND OTHER PLASMA PROTEINS DURING ABSTINENCE AFTER HEAVY ALCOHOL CONSUMPTION IN CHRONIC ALCOHOLICS. (Eng.) Wallerstedt, S.; Cederblad, G.; Korsan-Bengtsen, K.; Olsson, R. (Sahlgrenska sjukhuset, S-413 45, Goteborg, Sweden). *Scand. J. Gastroenterol.* 12(6): 649-655; 1977.

Variations in the concentrations of different liver-synthesized coagulation factors and other plasma proteins were studied in the withdrawal period after prolonged alcohol consumption in 19 male chronic alcoholics. The mean duration of chronic alcoholism was 11 ± 9 yr. The duration of the last debauch was 25 ± 30 days, and the final daily ethanol consumption during the last debauch was 354 ± 97 g. The coagulation factor levels were generally within the reference limits. In fact, the mean levels of factors VIII, IX, and XII were above the upper reference limits, the values being 153 ± 12%, 155 ± 14%, and 214 ± 21%, resp., in samples taken 1-3 days after the end of the debauch and 167 ± 19%, 152 ± 14%, and 198 ± 20%, resp., in samples taken 7-10 days after the end of the debauch. The variables reflecting the fibrinolytic system, i.e., lysis area (148 ± 5 and 149 ± 4 mm^2 on day 1-3 and day 7-10, resp.) and plasminogen (16.6 ± 1.0 and 18.2 ± 0.7 U/ml, resp.), were generally within the reference range, as were antithrombin III (97 ± 5% and 112 ± 5%, resp.) and $alpha_2$-macroglobulin (77 ± 3% and 83 ± 4%, resp.). The number of platelets was often low, and the mean value at day 1-3 was just above the lower reference limit (reference range 150-350 x 10^9/l; mean level in patients 163 ± 13 x 10^9/l). However, the platelet adhesiveness was generally within the reference range. During the 1st week, a significant increase occurred in the factor XI activity, the antithrombin III level, and the number of platelets, the levels being 127 ± 13% (increasing from 96 ± 7% at day 1-3, $p<0.01$), 112 ± 5% ($p<0.05$), and 239 ± 23 x 10^9/l ($p<0.01$) on day 7-10, resp. In samples taken on day 1-3, other plasma proteins were usually within the reference limits. During the 1st week, a significant increase occurred in prealbumin (from 0.224 ± 0.012 on day 1-3 to 0.255 ± 0.011 g/l on day 7-10, $p<0.01$), transferrin (from 2.30 ± 0.12 to 2.59 ± 0.11 g/l, $p<0.05$), and complement components C3 (from 0.70 ± 0.03 to 0.89 ± 0.05 g/l, $p<0.01$) and C4 (from 0.211 ± 0.013 to 0.247 ± 0.017 g/l, $p<0.01$). Orosomucoid, IgG, IgA, and IgM levels decreased significantly during the same period ($p<0.05$, $p<0.01$, $p<0.01$, and $p<0.05$, resp.). A significant correlation between the fibrinogen concentration and ESR was observed ($p<0.01$). The data indicate that the protein-synthesizing capacity of the liver is well preserved in the patients in spite of a decade of chronic alcoholism.

9156 LIVER INJURIES IN ALCOHOLICS. (Eng.) Abe, H.; Ikejiri, N.; Harada, K.; Ren-Pan, L.; Tanikawa, K.; Yokoyama, T.; *et al.* (Kurume Univ. Sch. Medicine, Kurume, 830, Japan). *Kurume Med. J.* 25(1):53-64; 1978.

The relationship of alcohol consumption to clinical findings, laboratory data, and liver histology was studied in 174 alcoholics. Histologically, there were 51 patients with minimal liver changes (M), 46 with fatty liver (F), 19 with fibrosis (Fi), 21 with alcoholic hepatitis (A), and 37 with cirrhosis (C). Alcoholic hyalin was seen in 4 patients with A and in 10 patients with C. Hepatomegaly was found in 108 patients, jaundice in 64, and esophageal varices in 13. Hepatomegaly was significantly ($p<0.025$) greater in patients with A and C than with M. The SGOT values were significantly higher ($p<0.005$) in patients with F, A, and C than with M and Fi. There was a significant ($p<0.001$) correlation between the SGOT level and both the lactate dehydrogenase (LDH) and creatinine phosphokinase (CPK) levels. SGPT values were also significantly higher in patients with A ($p<0.005$), F ($p<0.025$), and C ($p<0.05$) than with M and Fi. The SGOT/SGPT ratio was elevated in all groups. The SGPT level was also markedly elevated in all groups and was significantly higher in patients with A ($p<0.005$), Fi ($p<0.01$), and F ($p<0.025$) than with M. The serum alkaline phosphatase level in patients with C was significantly higher ($p<0.05$) than in those with M. Compared with M, elevations in total serum bilirubin level, in the rate of indocyanin green retention, and in the serum gammaglobulin level in patients with A and C were significant ($p<0.025$, <0.05, and <0.05, resp.) as was the lowering of the serum albumin level ($p<0.05$). Total serum cholesterol level was significantly ($p<0.05$) lower in patients with C than with M. The serum malonic dialdehyde (MDA) levels were elevated in all groups. Hepatitis B surface antigen was positive in 11.5% of the cases, indicating involvement of the hepatitis virus.

No significant changes in age and duration distribution were found among the groups and, while no significant differences in the daily and total alcohol consumption were observed, the trend was for patients with Fi to have a long duration and relatively small quantity of alcohol intake and for patients with C to have a long duration and a large quantity of alcohol intake. The authors conclude that while the amount and duration of alcohol intake is closely related to cirrhosis, other factors must be considered in the pathogenesis of alcoholic liver disease. [illegible] h average MDA values recorded sug[illegible] of lipoperoxide in the development [illegible] liver injuries.

9157 CLASSIFICATION AND FREQUENCY OF ALCOHOLIC LIVER DAMAGE. (Ger.) Roschlau, G. (Medizinischen Akademie "Carl Gustav Carus," Fetscherstr. 74, DDR-8019 Dresden, E. Germany). *Dtsch. Gesundheitsw.* 32(16):721-724; 1977.

9158 INVESTIGATION OF THE CELLULAR IMMUNE REACTIVITY IN ALCOHOLIC LIVER DAMAGE. (Ger.) Otto, L.; Nilius, R.; Busse, H. J.; Zipprich, B. (Klinikum Krollwitz, Ernst-Grube-Strasse, PSF 63, DDR-401 Halle [Saale], E. Germany). *Z. Gesamte Inn. Med.* 32(12):271-274; 1977.

9159 DEVELOPMENT OF TEMPORARY ASCITES IN ALCOHOLIC HEPATITIS. (Ger.) Poegel, K. (Stadtkrankenhaus Dresden-Neustadt, Industriestr. 40, DDR-8023 Dresden, E. Germany). *Dtsch. Gesundheitsw.* 32(30):1413-1415; 1977.

See also, 8818, 8828, 9079, 9165, 9171, 9233.

LIVER AND BILIARY TRACT

Cirrhosis

9160 CHANGES IN THE ACID-BASE EQUILIBRIUM IN LIVER CIRRHOSIS FOLLOWING PORTASYSTEMIC ANASTOMOSIS. (Ita.) Conte, G.; Semplicini, A. (Clinica Medica Generale II, Universita, Padua, Italy). *Recent. Prog. Med. (Roma)* 63(5):539-546; 1977.

Disturbances in acid-base equilibrium in hepatic cirrhosis induced by portasystemic anastomosis (respiratory alkalosis, metabolic alkalosis, and metabolic acidosis) are reviewed. The primary contributing factor to respiratory alkalosis after bypass surgery is the abnormally high serum level of ammonia, which stimulates the respiratory center and produces hyperventilation. The increase in serum ammonia is only in part lessened by a hypoprotein diet and by the use of antibiotics. Deteriorated liver functions also account for the increase in serum ammonia level as well as in the blood levels of lactate and pyruvate (probably further elevated by the increased activity of phosphofructokinase in an alkaline environment) and contribute to elevated levels of estrogen and progesterone. The respiratory alkalosis following bypass surgery can have secondary manifestations: reduction of the flow of blood to the brain (with symptoms of hyperammonemic encephalopathy), tetany and paresthesia due to decreased serum Ca levels. Respiratory alkalosis can be treated with chlorohydrated arginine. The most important cause of metabolic alkalosis after anastomosis is the depletion of potassium levels caused by the hypoprotein diet, impaired liver functions, and diuretic treatments. Diuretic treatments produce hyperaldosteronism because the loss of Na^+ and Cl^- contracts the extracellular volume and activates the renin-angiotensin system. The ultimate effects of the increase in aldosterone are increased elimination of K^+ and H^+, increased tubular reabsorption of HCO_3^-, diminished reabsorption of NaCl, and passage of extracellular H^+ into cells. Patients who have undergone anastomosis should be given KCl to decrease the incidence of postoperative metabolic alkalosis. Metabolic acidosis is a less frequent but more serious occurrence, caused by an increase in the production of hydrogen ions due to increased blood levels of lactate and pyruvate that result from profound derangements in carbohydrate metabolism. Decreased renal elimination of H^+ and an increase in H^+ concentration via the i.v. administration of acidic solutions also contribute to metabolic acidosis. If metabolic acidosis occurs after anastomosis, the use of acidic venous solutions and furosemide should be avoided.

9161 ALPHA$_1$-ANTITRYPSIN DEFICIENCY--A LITERATURE REVIEW AND A CASE REPORT OF A PA-

TIENT WITH CHRONIC OBSTRUCTIVE AIRWAYS DISEASE AND CIRRHOSIS. (Eng.) Coughlin, G. P.; van Deth, A. G.; Ey, G. T.; Grant, A. K. (Queen Elizabeth Hosp., Woodville, South Australia 5011, Australia). *Aust. N.Z. J. Med.* 7(4):400-403; 1977.

The case of a 52-yr-old man with alpha$_1$-antitrypsin deficiency (consistent with a state homozygous for the PiZ allele) is presented, and the literature concerning such cases is reviewed. The patient presented with a 12-yr history of increasing dyspnea on exertion and a productive cough. Clinical examination revealed severe obstructive deficit, panlobular emphysema, right ventricular hypertrophy, and an alpha$_1$-antitrypsin level of 50 mg/dl. Over the next 6 yr, he developed right heart failure, collapse of the thoracic vertebrae, renal failure, and mixed hepatitic-cholestatic liver function abnormalities (hepatitis B surface antigen was persistently negative). Postmortem examination revealed severe panlobular emphysema, extensive bullous changes throughout the lungs, patchy bronchopneumonia, mild fibrinous pleuritis, small amounts of thrombo-embolic material and occasional bone marrow emboli in the peripheral pulmonary arteries, marked right ventricular hypertrophy and dilatation, mild ischemic fibrosis of both ventricles, established micronodular cirrhosis with scattered areas of fatty change, numerous eosinophilic spherical intracytoplasmic PAS-positive and diastase resistant hyaline bodies in the hepatocytes, numerous intracytoplasmic inclusions bound by rough endoplasmic reticulum, mild vacuolar nephropathy, marked hypertrophy of the median lobe of the prostate, and slight dilatation of the ureters (suggesting bladder-neck obstruction as a cause for renal failure).

9162 CHOLESTATIC LIVER CIRRHOSIS WITH AUTOIMMUNE PHENOMENA IN IDENTICAL TWIN GIRLS. (Ger.) Kirchhof, M.; Dammermann, R.; Lindner, H.; Berg, P. A.; Kloppel, G. (DRK-Krankenhaus, Beim Schlump 84, 2000 Hamburg 13, W. Germany). *Therapiewoche* 28(6):1105-1106; 1978.

Cholestatic liver cirrhosis with portal hypertension and esophageal varices was found in two 45-yr-old identical twin sisters. Mitochondrial antibodies were sometimes present during the early stage of the disease, never during the final stage. The IgM level was not increased. A combination of antinuclear factors of nucleolar type, antibodies to smooth muscles, and cytoplasmic antibodies to kidneys, stomach, and thyroid cells were found. Nuclear antibodies were found in 4/12 children of the identical twins, but there were no manifest hepatopathies. The father of the identical twins had had liver disease (not specified) and pernicious anemia. The findings justify antibody tests in family members of patients with autoimmune chronic liver diseases.

9163 WHOLE GUT IRRIGATION WITH ISOTONIC MANNITOL SOLUTION IN TREATMENT OF GASTROINTESTINAL BLEEDING DUE TO CIRRHOSIS: A CONTROLLED STUDY. (Fre.) Hecketsweiler, P.; Colin, R.; Ouvry, D.; Galmiche, J. P.; Paillot, B.; Geffroy, Y. (Hopital Charles-Nicolle, F 76038 Rouen Cedex, France). *Gastroenterol. Clin. Biol.* 2(2):145-152; 1978.

Whole-gut irrigation with an isotonic mannitol solution (40 g/l) containing 17 mM NaCl, 13 mM KCl, and neomycin sulfate (250 mg/l) was used to treat gastrointestinal bleeding in 29 patients with liver cirrhosis, and the results were compared with those obtained in 30 age-, sex-, and symptom-matched controls treated without gut irrigation. All patients had portal hypertension secondary to alcoholic cirrhosis, and blood was present in the upper gastrointestinal tract although the hemorrhage source (such as esophageal varices or gastroduodenal lesions) was not always known. After fibroscopy examination and balloon tamponade insertion for hemorrhage, the control patients received gastric lavage and aspiration, neomycin (0.75 g), and lactulose (50 ml) and the study group received mannitol solution (5 l, 33 ml/min perfusion). Twelve hours later and every 12 hr during the period the patient was bleeding, 0.75 g neomycin and 50 ml lactulose were administered. In the treatment group, only one patient developed hepatic encephalopathy (HE) in the 24 hr after treatment, whereas in the control group, five developed HE ($p=0.08$). Five and nine patients in these two groups, resp., had HE before the study; regression of HE occurred in four of five in the first 24 hr after gut irrigation but in none of the control patients ($p=0.01$). After 24 hr of the study, 2/29 and 13/26 patients in the treatment and control groups, resp., had HE ($p<0.001$). In the control group, 3 patients died in the first 12 hr of the study and 10 more died of hemorrhage or HE within 12 days of admission. In the treatment group, only four patients died within the 12-day period ($p<0.02$). The duration of hemorrhage was not significantly different in the two groups, but the number of units of blood required to maintain a hematocrit of 35% was significantly less ($p<0.02$) in the treatment group. Of 18 controls and 22 treatment patients who entered the study without ascites and who were alive on the 7th day of the study, 8 and 2 patients, resp., developed ascites ($p<0.05$). Gut irrigation with an isotonic mannitol solution appears to prevent HE in cirrhotic patients with gastrointestinal hemorrhage and to improve prognosis.

9164 GASTRIC NARROWING WITH INTRALUMINAL FILLING DEFECT IN A 55-YEAR-OLD WOMAN. (Eng.) Margolies, M. N.; Caldabini, J. J.; McNeely, B. U. (Massachusetts General Hosp., Boston, MA). *N. Engl. J. Med.* 298(23):1301-1307; 1978.

A case record is presented concerning a woman with a life-long history of portal hypertension due to neonatal portal vein thrombosis who was admitted to the hospital with progressive epigastric burning pain, which was relieved promptly by vomiting. Seven months before admission, a radiologic abnormality in the gastric antrum had been detected after an episode of upper gastrointestinal bleeding. This had been treated with antacid, diazepam, atro-

pine, and ferrous sulfate. After 4 months she began to experience the abdominal pain for which she was finally hospitalized. The patient had been under emotional stress. Laboratory findings were normal except for occult blood in the stool. Radiologic examination demonstrated gallstones and a stenotic lesion in the antrum that had progressed over a period of several months with the subsequent appearance of an intraluminal filling defect. Among the diseases considered and rejected in the differential diagnosis were Crohn's disease, benign gastric tumors, carcinoma of the gastric antrum, and lymphomas of the stomach. Gallstones were considered to be an incidental finding, and diagnosis was made of a cicatricial peptic ulcer with obstruction due to a foreign body retained in the stomach, a gastric bezoar. The bezoar was confirmed and removed endoscopically. It measured 2 x 1.5 x 1.1 cm, and was ovoid, firm, and externally greenish-black, with a gray interior. An irregular central cavity was filled with saprophytic bacteria and fungi. Microscopic examination showed it to be a phytobezoar, consisting of granular plant parenchyma and a few plant vascular elements.

9165 MALABSORPTION OF VITAMIN D IN MAN AND RAT WITH LIVER CIRRHOSIS. (Eng.) Meyer, M.; Wechsler, S.; Shibolet, S.; Jedwab, M.; Harell, A.; Edelstein, S. (Dept. Internal Medicine D, Ichilov Hosp., 6 Weizmann St., Tel Aviv, Israel). *J. Mol. Med.* 3(1):29-37; 1978.

To localize the defect in vitamin D metabolism leading to the reduced circulating level of 25-hydroxyvitamin D observed in liver cirrhosis, vitamin D metabolism and absorption were studied in four patients with alcoholic or postnecrotic liver cirrhosis, three control patients, and 20 male Wistar rats with carbon tetrachloride-induced cirrhosis. In neither cirrhotic patients nor cirrhotic rats was there impairment of the conversion of i.v injected tritiated vitamin D to 25-hydroxyvitamin D. However, the level of cholecalciferol in the blood of both patients and cirrhotic animals that received tritiated cholecalciferol intragastrically was significantly lower at all times studied than that in the controls ($p<0.01$). It appears that impaired absorption of dietary vitamin D-3 may contribute to or be one of the etiologic factors in reducing the serum 25-hydroxyvitamin D levels in liver cirrhosis.

9166 FAMILIAL LIVER CIRRHOSIS IN PSEUDOHYPOPARATHYROIDISM IN YOUNG CHILDREN. (Ger.) Thanner, F.; Kuhner, U.; Pfeifer, U. (Universitaets-Kinderklinik Wurzburg, Joseph-Schneider Strasse 2, 8700 Wurzburg, W. Germany). *Dtsch. Med. Wochenschr.* 103(12):519-523; 1978.

The appearance of pseudohypoparathyroidism (PHPT) in infancy has not been described previously, nor has the combination of PHPT and severe congenital liver disease. Two cases of this nature are described: two girls whose mother had PHPT developed PHPT with congenital liver damage. The first girl developed icterus on the fourth postnatal day, and was hospitalized at 2 months for persistent icterus and dystrophy. She was rehospitalized at 2 yr with hepatomegaly and splenomegaly, hypocalcemia, hyperphosphatemia, thrombocytopenia, and possible tetany. Laparoscopy showed postnecrotic hepatic cirrhosis with beginning portal hypertension. Histology of hepatic tissue demonstrated almost complete cirrhosis in some areas, areas of fibrosis, hyaline single cell necrosis, intracellular fat accumulation, and siderin present in parenchyma and stellate cells. Mesenchyme activity showed that the process was continuing. The child died at 4 yr following internal hemorrhage, despite treatment with calcium and vitamin D. The second girl, with prenatal dystrophy, was shown to have fatty liver deposits, isolated hyalin liver necrosis, and centrilobular multinuclear giant cells at birth. The course of the disease in this child was similar to that in her sister. Therapeutic administration of dihydrotachysterol, vitamin D, and calcium improved the state of the disease only transiently. Liver biopsy at 1.5 yr demonstrated the presence of "ectoplasmic vacuoles" by electron microscopy. The authors suggest that this combined disorder in calcium and liver metabolism may be the result of a genetic defect, as known metabolic or inflammatory cell diseases were excluded.

9167 ACUTE RENAL INSUFFICIENCY DUE TO NECROSIS OF THE RENAL MEDULLA IN CIRRHOTIC PATIENTS. (Fre.) Dequiedt, P.; Dequiedt, F.; Vanhille, P.; Raviart, B.; Lambert, P.; Mouton, Y.; *et al.* (Hopital G. Dron, 59208 Tourcoing, France). *Lille Med.* 21(8):674-678; 1976.

The frequency, clinical profile, differential diagnosis, pathology, and treatment of acute renal insufficiency due to medullary necrosis of the kidneys and associated with liver cirrhosis are reviewed, and clinical profiles of three male patients (ages 54, 45, and 47 yr, resp.) are given. Insufficiency, which occurs in 2% of cirrhotics, is usually of sudden onset with oliguria accompanied by a severe infection. Two of the three patients had prodromal symptoms: pain and fever in one case and urinary infection and hematuria in the others. All three patients had accompanying lumbar pain, but icterus was present in only two. Plasma creatinine and urea levels were elevated, and the Na^+/K^+ ratio was >1. Correction of the acid-base balance and administration of antibiotics were used to treat the patients; however, two died on the 11th and 12th days, resp., following onset of renal insufficiency. Pathology other than kidney necrosis (acute pancreatitis, hematological interstitial nephritis, and acute tubulonephritis) can be the cause of acute renal insufficiency in chronic liver cirrhosis. Acute renal insufficiency of an apparent organic cause accompanied by septicemic infection in a cirrhotic patient should indicate the diagnosis of kidney necrosis. Changes in the kidney circulation during liver cirrhosis lead to medullary necrosis and thrombosis.

9168 SERUM CONCENTRATIONS AND URINARY EXCRETIONS OF ZINC IN CIRRHOSIS, NEPHROTIC SYNDROME AND RENAL INSUFFICIENCY. (Eng.) Lindeman,

R. D.; Baxter, D. J.; Yunice, A. A.; Kraikitpanitch, S. (Veterans Admin. Hosp., 921 N.E. 13th St., Oklahoma City, OK 73104). *Am. J. Med. Sci.* 275(1): 17-31; 1978.

Serum Zn concentrations were measured in 12 patients with cirrhosis, 23 patients with nephrotic syndrome, and 13 patients with renal insufficiency to document the frequency of low serum Zn concentrations and to define the importance of urinary loss of Zn in the Zn deficiency commonly seen in these patients. Multiple correlations between the urinary zinc and protein excretions, and the patterns of binding between added radiozinc and serum and urine proteins determined by column chromatography and polyacrylamide gel electrophoresis were used to determine differences between the patients and 16 normal controls. The mean serum Zn concentrations were 90, 76, 66, and 49 µg/100 ml in normal, azotemic, nephrotic, and cirrhotic subjects, resp., the latter two values being significantly different ($p<0.01$) from the normal value. The mean 24-hr urinary Zn excretion was increased in the cirrhotic ($p<0.05$) and nephrotic patients relative to controls. In the azotemic group, the mean urinary Zn excretion was lower than in controls. No correlation ($r=0.097$) was found between serum Zn concentrations and urinary Zn excretions in the four groups combined, nor was there a significant relationship between serum Zn concentration and urinary protein excretion ($r=0.272$). There was a significant positive correlation ($r=0.340$, $p<0.05$) between serum Zn and albumin concentrations in the four groups combined but not for any individual patient group. In the nephrotic patients, there was a significant correlation between urinary Zn and protein excretions of ($r=0.431$, $p<0.05$). Sephadex G-200 gel chromatographic patterns for the sera and urine of controls and cirrhotic patients were similar except that, in the patients, peak II (containing albumins and prealbumins) was decreased. The patterns for the other two patient groups were less consistent, but both nephrotic and azotemic patients showed significant ($p<0.001$) increases in serum peak III (containing low molecular weight proteins, polypeptides, and amino acids) when compared with normal subjects. Polyacrylamide gel electrophoresis showed the mean percentage of radiozinc added to the serum that bound in the albumin peak was 95, 76, 47, and 63% in normal, cirrhotic, nephrotic, and azotemic patients, resp. The current study adds to the available data on Zn deficiency in cirrhotic, nephrotic, and azotemic patients but it remains unclear whether the low serum Zn concentrations are indicative of true clinical or subclinical Zn deficiency or simply reflect a decrease in available Zn-binding proteins or prealbumin.

9169 TUBULAR RENAL ACIDOSIS DURING LIVER CIRRHOSIS. (Ita.) Triolo, R.; Carelli, E.; D'Alessio, R.; Italiano, F. (I Divisione di Medicina Generale, Ospedale Martini, Torino, Italy). *Minerva Med.* 68(53):3627-3642; 1977.

To investigate the behavior of the acid-base equilibrium in cirrhosis of the liver and to determine the effects of this disease on tubular renal functions, blood and urine analyses were made in 23 patients with liver cirrhosis. SGOT, SGPT, LDH, alkaline phosphatase, protein, bilirubin, glucose, calcium, phosphorus, sodium, potassium, chlorine, ammonia, bicarbonate, creatinine, and pH levels were determined. Patients were divided into three groups: seven patients with plasma pH <7.37 (group 1), four patients with a loss of bicarbonate (group 2), and two patients with plasma pH <7.37 without a loss of bicarbonate (group 3). The plasma analyses showed that the bicarbonate level was low in all three groups, with levels in group 3 being the lowest. Ammonia levels were normal in group 3 and raised in groups 1 and 2. Sodium was normal in groups 2 and 3 and low in group 1. Phosphorus was normal in patients with acidosis and loss of bicarbonate but low in the other patients. The bilirubin level was high in groups 1 and 2. The urine analyses revealed that ammonia levels were normal in all three groups. The acidity was normal in groups 1 and 3 but low in group 2. The bicarbonate filtrates were reduced in groups 1 and 3 and in patients with tubular renal acidosis type II complete, but it was raised in patients with tubular renal acidosis type II incomplete. The percentage of reabsorbed bicarbonate was reduced in group 2. The loss of sodium via the urine was greatly augmented in all three groups. It was calculated that 18 of the patients had supercompensated metabolic acidosis, and 5 had normally compensated metabolic acidosis.

9170 ELECTIVE DECOMPRESSION OF ESOPHAGEAL VARICES BY DISTAL SPLENO-RENAL BYPASS USING WARREN'S METHOD. (Fre.) Mosimann, R.; Loup, P. (Centre Hospitalier Universitaire Vaudois, 1011 Lausanne, Switzerland). *J. Chir. (Paris)* 114(3): 175-184; 1977.

The results of treatment by splenorenal bypass in 22 patients with hemorrhaging esophageal varices related to liver cirrhosis and portal hypertension are presented. The cirrhosis was due to alcohol in 12 patients, to hepatitis in 8, to primary biliary cirrhosis in 1, and to alcoholic steatosis in 1. A termino-lateral anastomosis of the splenic and renal veins was performed in 12 patients (Warren's original method), and a termino-terminal anastomsis with ligature of the renal vein before the branching of the suprarenal and gonadal veins was performed in 10 patients. Two patients died postoperatively, one of hemorrhage from a duodenal stress ulcer and the other in hepatic coma following two reoperations for thrombosis of the shunt. Stenosis of the shunt occurred in another patient who survived but required portacaval anastomosis to control massive new hemorrhaging. Retrogastric lymphocele requiring drainage complicated the postoperative course of a fourth patient. Barium transit studies carried out in 17/20 surviving patients indicated disappearance or decrease of the esophageal varices in 13. In addition to the two patients who died postoperatively and the survivor who rebled, two other survivors hemorrhaged again, one at 6 months (autopsy revealed thrombosis

of the portal vein and erosive gastritis, but the shunt was patent) and one after many months following an episode of alcohol abuse. No other patients rebled. In addition to this one late death, four other patients died during the follow-up period, all due to hepatic insufficiency. Twelve patients who had been followed for 6 months to 7 yr (average 2.5 yr) were still alive. One of the survivors had temporary encephalopathy in the early postoperative period. This operation is useful in treating portal hypertension when there is satisfactory portal blood flow, but it is contraindicated in the case of greatly reduced or inverted portal flow or in case of ascites or marked stasis of the splanchnic or retroperitoneal lymphatic network.

9171 THE CHANGING PATTERN OF LIVER DISEASE IN SOUTH AFRICAN BLACKS. (Eng.) Isaacson, C. (South African Inst. Medical Res., Johannesburg, South Africa). *S. Afr. Med. J.* 53(10):365-368; 1978.

9172 EXTRAHEPATIC ELIMINATION OF GALACTOSE IN MAN [Abstract]. (Eng.) Ranek, L. (Rigshospitalet, Copenhagen, Denmark). *Digestion* 16(4): 329; 1977.

9173 THE USE OF LACTULOSE IN PORTAL-SYSTEMIC ENCEPHALOPATHY. (Eng.) Matthews, S. J. (Hartford Hosp., Hartford, CT). *Conn. Med.* 42(2): 96-97; 1978.

See also, 8798, 8828, 8832, 9079, 9080, 9081, 9086, 9090, 9095, 9096, 9099, 9135, 9149, 9159, 9235.

LIVER AND BILIARY TRACT

Gallbladder and Biliary Tract Diseases

9174 WHEN CHOLECYSTECTOMY? (Eng.) Naitove, A. (Dartmouth Medical Sch., Hanover, NH). *Hosp. Pract.* 13(6):121-128; 1978.

Factors that should be considered in determining which patients should undergo cholecystectomy are discussed. Acutely ill patients with an increased risk of operative mortality should avoid cholecystectomy. In patients with classic symptoms of chronic cholecystitis and radiologically demonstrated gallstones or a nonfunctioning gallbladder, elective cholecystectomy should be carried out provided there are no medical contraindications (including those imposed by old age). Previous studies have shown that severe complications (acute cholecystitis, cholangitis, jaundice, pancreatitis) occur in these patients when surgery is not carried out. Observation of asymptomatic or very mildly symptomatic patients has shown that gallbladder disease often does not progress in a predictable way from asymptomatic to mildly symptomatic to severely symptomatic to complicated; many patients are symptom-free from the initial diagnosis by cholecystography until the day a complication appears. The author's experience suggests that patients with symptoms of cholelithiasis that cannot be ascribed to any other cause, but which cannot be explained by any diagnostic procedure, should undergo surgery for suspected occult gallstone disease. Thus, elective cholecystectomy should be carried out in (a) patients with symptoms and radiological evidence of gallstones, (b) asymptomatic patients with radiologic evidence of gallstones, and (c) patients with symptoms but without radiologic evidence of disease. Since acute attacks of cholecystitis will subside in about 75-80% of patients, the primary test is to identify the optimal time for surgical intervention. In patients in whom advanced age is not a factor, in whom the diagnosis of acute cholecystitis is relatively certain, and for whom cholecystectomy is the inevitable choice of definitive treatment, operation should often be performed after 2 or 3 days of hospitalization and medical management. For older patients who may have other debilitating diseases or for those in whom further diagnostic workup is indicated, elective surgery 6-8 weeks later may be preferable. In patients in whom symptoms either do not improve or worsen during the first 48 hr after the onset of symptoms, emergency surgery is indicated.

9175 BACTERIAL FLORA OF THE SMALL INTESTINE AND BILE ACID METABOLISM IN PATIENTS WITH HEPATICO-JEJUNOSTOMY ROUX-EN-Y. (Eng.) Nielsen, M. L.; Justesen, T.; Lenz, K.; Nielsen, O. V.; Jensen, S. L. (31 Sofievej, DK-2900 Hellerup, Denmark). *Scand. J. Gastroenterol.* 12(8):977-982; 1977.

To determine whether ascending biliary tract infection from an intestinal blind loop with bacterial overgrowth could result in the polymicrobial flora seen in the bile of patients with hepaticojejunostomy, duodenal and jejunal bacterial flora and bile acid metabolism were investigated in 14 patients with Roux-en-Y hepaticojejunostomy. Duodenal aspirates were normal in

seven patients, whereas abnormal bacterial flora were found in the other seven. The jejunal aspirate, taken in the most proximal part of the jejunum at the site of the entero-entero anastomosis, was normal in only one patient. The difference between the number of abnormal duodenal and abnormal jejunal aspirates was significant ($p<0.05$). Total bile acid concentrations in jejunal aspirates at the anastomosis were low in half the patients (<5 mM). Free (deconjugated) bile acids were found in only one patient, and only trace amounts were present. Free bile acids and an elevated glycine/taurine ratio (6.8) were found in one patient each. The 1-^{14}C-cholyl-glycine breath test was abnormal (>3% of dose expired as $^{14}CO_2$) in five patients, and abnormal ^{14}C output in stools (>5%) was observed in three patients. It is concluded that Roux-en-Y hepaticojejunostomy causes abnormal bacterial flora in the proximal jejunum of almost all patients, whereas alterations in bile acid metabolism are less frequent.

9176 INVESTIGATIONS ON ALBUMIN EXCRETION BY THE LIVER INTO THE BILE. I. POSTOPERATIVE ALBUMIN EXCRETION INTO THE T-DRAINAGE BILE. (Ger.) Rogos, R. (Kreiskrankenhaus, Leipziger Str. 55, DDR-74 Altenburg, E. Germany). *Dtsch. Z. Verdau. Stoffwechselkr.* 38(1):21-26; 1978.

The albumin level was determined by radial immunodiffusion and disc electrophoresis in 134 T-drain bile samples from 62 patients who underwent surgery for cholelithiasis, choledocholithiasis, cholecystitis, and cholangitis. The albumin concentration in the bile of these patients averaged 83.3 ± 61.2 mg/100 ml on postoperative day 1, 37.2 ± 34.7 mg/100 ml on day 3, and 23 ± 22.8 mg/100 ml on day 5. The albumin excretion rate was 228.8 ± 191.1 mg/day on day 1, 118.0 ± 96.7 mg/day on day 3, and 86 ± 86.2 mg/day on day 5. Albumin accounted for 41.8% of the total protein on day 1, for 39% on day 3, and for 40.8% on day 5. Compared with a normal albumin catabolism of 12 g/day, the postoperative albumin loss in the T-drain bile is negligible.

9177 EXCRETION OF METRONIDAZOLE IN HUMAN BILE: INVESTIGATIONS OF HEPATIC BILE, COMMON DUCT BILE, AND GALLBLADDER BILE. (Eng.) Nielsen, M. L.; Justesen, T. (31 Sofievej, DK-2900 Hellerup, Denmark). *Scand. J. Gastroenterol.* 12(8):1003-1008; 1977.

To investigate the possible value of metronidazole (MNZ) in the treatment of biliary tract infections, MNZ excretion after i.v. injection (500 mg) was studied during surgery in 11 patients without biliary tract disease, 17 patients with gallstones with a normal common duct and normal gallbladder function, 22 patients with gallstones with a normal common duct and loss of gallbladder function, and 5 patients with common duct stones, common duct obstruction, and jaundice. Excretion of MNZ (2 g, p.o.) in hepatic bile was also determined in three other patients following surgery; MNZ appeared rapidly in hepatic bile, and throughout the 17-hr observation period almost identical concentrations of MNZ were found in serum and hepatic bile. In the patients with a normal biliary tract given an i.v. MNZ injection, significantly higher concentrations of MNZ were found in gallbladder bile than in serum ($p<0.01$). In patients with gallstones and preserved gallbladder function, no differences were found between the MNZ concentrations in the serum, common duct bile, and gallbladder bile. In patients with gallstones and loss of gallbladder function, no differences were found between the MNZ concentrations in the serum and in common duct bile, whereas the MNZ concentration in gallbladder bile was significantly lower than that in the serum and common duct bile ($p<0.01$). When MNZ concentrations in gallbladder bile were compared, significant ($p<0.01$) differences were found between all three groups of patients: the highest concentrations were found in normal gallbladders and the lowest concentrations were found in nonfunctional gallbladders. In 9 of 14 patients with a blocked cystic duct, no MNZ was detected in gallbladder bile and in the remaining 5 patients, a concentration of 43-63% of the MNZ in serum was found. Thus, MNZ concentrations in gallbladder bile were significantly ($p<0.01$) lower than MNZ concentrations in both serum and common duct bile, whereas no significant difference was found between serum and common duct bile. In eight patients with a patent cystic duct, no significant differences were found between MNZ concentrations in serum, common duct bile, and gallbladder bile. Common duct bile MNZ concentrations in the patients with common duct obstruction varied from 56% to 99% of the corresponding serum MNZ concentration. I.v. MNZ would be useful in the chemotherapy of cholangitis.

9178 CARCINOMA OF THE PAPILLA OF VATER: A REVIEW OF FIFTY-SEVEN CASES. (Eng.) Schlippert, W.; Lucke, D.; Anuras, S.; Christensen, J. (Dept. Medicine, Univ. Iowa, Iowa City, IA 52242). *Am. J. Surg.* 135(6):757-762; 1978.

Experience with 57 cases of carcinoma of the papilla of Vater are reviewed to determine the relative success of various kinds of surgery and to define those clinical characteristics that suggest the diagnosis and that correlate with survival, palliation, and cure. The relative frequencies for both pancreatic and papillary malignancies increased little since 1940, with approximately 13-16 cases of pancreatic malignancy occurring for each case of papillary tumor. The most common presenting complaints were weight loss in 94% and jaundice in 91%. Hepatic enlargement was discovered on physical examination in 64%. Of 47 upper gastrointestinal contrast studies, 26 were interpreted as normal. The 21 abnormal results included abnormalities of the second part of the duodenum in 12 patients, extrinsic pressure on the stomach or duodenum in 5, the "inverted three" sign in 1, and partial duodenal obstruction by an annular lesion in 1. Endoscopic retrograde cholangiopancreatography confirmed the diagnosis in the

two patients in whom it was carried out. Percutaneous transhepatic cholangiography was performed in three patients and was abnormal in all, but celiac angiography was noncontributory in the two patients in whom it was performed. Local resection was carried out in seven patients. One patient is alive and well 12 yr after resection, and another died during the immediate postoperative period; the mean survival in the remaining cases was 21.3 ± 2.0 months (range 1.5–58 months). No metastases were found at the time of resection in any of these seven cases. A bypass procedure was performed initially in 16 patients. Metastases were noted in six cases. There were three operative deaths, and the mean survival of the remaining patients was 13.1 ± 1.3 months (1.5–41 months). A Whipple operation was performed initially in 31 patients, and metastases were found in 9 cases. There were seven operative deaths. Three patients are still alive 6, 6, and 84 months postoperatively. Two patients died of unrelated diseases. Mean survival excluding two current survivors whose survival is less than the median was 24.1 ± 1.2 months (11–53 months). When patients with metastases were considered separately, those who had a Whipple operation survived significantly longer than those who had a bypass operation ($p<0.05$). The Whipple procedure is probably the better operation for carcinoma of the papilla of Vater. Local resection in the absence of metastases offers a chance for cure and a survival rate that do not differ from those of the Whipple operation, although 50% of the cases in this series who underwent resection needed subsequent palliative surgery.

9179 CARCINOMA ARISING IN A CONGENITALLY DILATED BILIARY TRACT: REPORT OF A CASE AND REVIEW OF THE LITERATURE. (Eng.) Kagawa, Y.; Kashihara, S.; Kuramoto, S.; Maetani, S. (Tenri Hosp., Tenri, Nara, Japan). *Gastroenterology* 74(6): 1286–1294; 1978.

The case of a patient with carcinoma arising in a congenitally dilated biliary tract is reported, and the 47 cases in the world literature are reviewed. The patient was a 57-yr-old woman with a 5-yr history of episodes of characteristic symptoms of cholangitis. Endoscopic retrograde cholangiopancreatography disclosed a markedly dilated common duct, 20 mm in diameter in the intrapancreatic portion and 40 mm in the supraduodenal portion. The upper part of the common duct and the gallbladder were not fully visualized because of incidental incompetence of Oddi's sphincter. Percutaneous transhepatic cholangiography revealed dilated intrahepatic ducts and obstruction near the confluence of the hepatic ducts. Selective angiography revealed irregularity and tumor stain of the branches of the hepatic artery, which derived from the superior mesenteric artery. At surgery, the gallbladder was distended, and the common duct was dilated, having a diameter of 50 mm; neither contained stones. A papillary tumor was found filling the common bile duct lumen. Biopsy of the lesion confirmed papillary adenocarcinoma. The common duct, common hepatic duct, gallbladder, and regional lymph nodes were resected, and Roux en-y hepaticojejunostomy was performed at the bifurcation of the hepatic ducts. The patient has been asymptomatic for 30 months postoperatively. Drip infusion cholangiography at 4 and 10 months after surgery revealed dilated intrahepatic ducts. There was no evidence of obstructive jaundice, indicating that the dilatation of the intrahepatic ducts was congenital and not secondary to obstruction. A review of the literature suggests that the association of carcinoma with congenital biliary ductal dilatation is more frequent than is commonly recognized. Carcinoma may arise in either the extrahepatic or intrahepatic ducts. Primary excision of choledochal cysts is advisable, to prevent the risk of later malignant change.

9180 LIPOPROTEIN METABOLISM IN CHOLESTASIS. (Ger.) Patsch, W. (Medizinische Universitatsklinik, Anichstrasse 35, A-6020 Innsbruck, Austria). *Acta Med. Austriaca* 4(Special Issue): 86–87; 1977.

The relationships between lipoprotein-X (LP-X) and lecithin cholesterol acyltransferase (LCAT) activity were studied in three patients following the surgical removal of bile flow obstruction. The LCAT activity reached 3–4 times the normal value on the 4th postoperative day, while the concentration of LP-X_2, decreased rapidly. On the 6th postoperative day, the concentration of LP-X, also decreased rapidly. When LP-X_1 and LP-X_2 were incubated separately in normal plasma at 37 C, the half-life of LP-X_1 was 24 hr and that of LP-X_2 9.5 hr; the difference may be due to different apolipoprotein levels in the two compounds. During the reduction of the LP-X concentration in the medium, there appeared a high-density lipoprotein (HDL_2), which suggests that the decomposition of LP-X is mediated by an acyltransferase. The findings indicate the existence of a relationship between LP-X and LCAT.

9181 INDUCTION OF BILE ACID GLUCURONIDE FORMATION IN CHILDREN WITH INTRAHEPATIC CHOLESTASIS. (Eng.) Frohling, W.; Stiehl, A.; Czygan, P.; Liersch, M.; Kommerell, B.; Rotthauwe, H. W.; *et al.* *In: Bile Acid Metabolism in Health and Disease. Proceedings of the IV Bile Acid Meeting held at the Hilton Hotel, Basel, Switzerland, October 11-12, 1976.* (Baltimore: University Park Press): 101–104; 1977.

The effect of phenobarbital on intrahepatic cholestasis was reevaluated in three children with intrahepatic cholestasis, with special regard to a possible induction of bile acid glucuronide formation. The patients were treated with 0.1–0.14 g phenobarbital daily for at least 14 days. Phenobarbital caused an improvement of cholestasis, the disappearance of jaundice, and a decrease in serum bile acid concentrations of 60% (from 119 to 41 µg/ml) in all patients; nonsulfated, nonglucuronidated bile acids and bile acid sulfates were markedly reduced, whereas the decrease of glucuronides was much smaller

(glucuronides accounted for 14% of the serum bile acids before treatment and for 27% after treatment). Biliary bile acid excretion increased by 47% (78% for nonsulfated, nonglucuronidated bile acids, 62% for sulfates). Bile acid glucuronides increased by 122%, accounting for 12% of the bile acids before treatment and for 18% after treatment. The percentage of glucuronides of the different bile acids in bile ranged from 12-29% before treatment and was increased by phenobarbital for all bile acids investigated. Remarkably high rates of bile acid glucuronides, accounting for 40-60% in two children, were found for Δ-5-cholenic acid and lithocholic acid after treatment, suggesting a correlation between extensive glucuronidation and the disappearance of these highly toxic bile acids after phenobarbital treatment. Bile acid sulfate excretion in bile decreased after phenobarbital; 25% of bile acids were sulfated before treatment and 6% after treatment. Urinary bile acid excretion decreased by 45% after treatment, due to the lowered serum bile acid concentrations. Sulfated and nonsulfated, nonglucuronidated bile acids were similarly reduced, whereas the excretion of glucuronides increased by 136%. The alteration in the bile acid pattern after phenobarbital, with an increase in the percentage of serum, bile, and urinary bile acid glucuronides and an increase in the absolute amounts of glucuronides excreted in bile and urine, strongly suggests an increased formation of bile acid glucuronides after phenobarbital treatment.

9182 CONGENITAL CYSTIC DILATATION OF THE INTRA- AND EXTRAHEPATIC BILIARY TRACT. (Fre.) Bloch, P.; Dimitrov, N.; Eidesheim, D.; Clavert, A.; Christmann, D.; Kim, K.; *et al.* (Service de Radiologie, C.H.U. F67005 Strasbourg Cedex, France). *J. Radiol. Electrol. Med. Nucl.* 58(12):839-840; 1977.

A case of a cystic dilatation of the intra- and extrahepatic biliary tract in a 12-yr-old girl is reported. She was hospitalized for a painful abdomen syndrome that was diagnosed as acute pancreatitis. Her past medical history included an episode of icterus at the age of 4 yr and two other hospitalizations for painful abdomen. No abdominal mass was palpated. X-rays showed an enlargement of the retrogastric space and compression of the stomach. I.v. cholangiography revealed the cystic dilatation of the biliary tract. At surgery, the latter diagnosis was confirmed and two cysts were found, one a 5-cm cyst of the gallbladder and the other a cyst of the common hepatic duct. The pancreas was hard and enlarged, which accounted for the enlargement of the retrogastric space. The long-term results of surgical treatment have been excellent.

9183 MIRIZZI'S SYNDROME AS A SPECIAL FORM OF OBSTRUCTIVE JAUNDICE. (Ger.) Heil, T.; Belohlavek, D. (Zentrum fur Operative Medizin der Universitat, Steinhovelstrasse 9, D-7900 Ulm/Donau, W. Germany). *Chirurg* 49(1):57-59; 1978.

Mirizzi's syndrome (isolated stenosis of the hepatic duct) was diagnosed in three patients. Recurrent fever and right epigastric pains were the first symptoms. Two patients had jaundice. Endoscopic retrograde cholangiography was performed in two cases; it demonstrated Mirizzi's syndrome in both. Gallstones were found in the cystic duct in two patients and in the gallbladder in the third.

9184 HEPATIC NECROSES AFTER INFUSION CHOLANGIOGRAPHY. (Ger.) Winckler, K. (Abteilung fur Gastroenterologie und Stoffwechselkrankheiten, Medizinische Universitatsklinik, Robert-Koch-Str. 40, 3400 Gottingen, W. Germany). *Dtsch. Med. Wochenschr.* 103(10):420-424; 1978.

Ten episodes of massive increase of transaminase levels accompanying liver necrosis were observed in seven patients (1 each with biliary tract disease, recurrent pancreatitis, cholelithiasis, hyperlipemia and minimal toxic liver damage, pyelonephritis, arteriitis, and pseudolupus erythematosus) following infusion of megluminioglycamide (Biligram; 100 ml in 5 cases, 200 ml in 2). In six patients, there was an increased alkaline phosphatase level without jaundice before the investigation, and a slight increase in the transaminase levels in three. After Biligram infusion, there was an abrupt increase in the SGPT level (range 98-2,202 U/l), and a lesser increase in the SGOT level, but the alkaline phosphatase level showed no major change. Abdominal pain, fever, erythema, or conjunctivitis were seen in three patients. Histologically, all patients showed centrilobular necrosis. Transaminase levels should be checked 2 days after infusion cholangiography; in patients showing definite increase, reexposure to the contrast agent should be avoided.

9185 BILE-DUCT OBSTRUCTION IN A 39-YEAR-OLD MAN. (Eng.) Burhenne, H. J.; Galdabini, J. J. (Massachusetts General Hosp., Boston, MA). *N. Engl. J. Med.* 297(19):1054-1059; 1977.

The case of a patient with bile duct obstruction and a discussion of the differential and clinical diagnosis are presented. The patient developed jaundice 1 week after receiving halothane anesthesia for herniorrhaphy. An ultrasound study demonstrated distended intrahepatic bile ducts. Upper gastrointestinal series and hypotonic duodenography helped rule out an obstructing pancreatic lesion. Endoscopic retrograde cholangiopancreatography revealed complete obstruction of the common hepatic duct 1 cm proximal to the insertion of the cystic duct. The contrast column at the point of obstruction showed a smooth, conical narrowing. Distal to the obstructed area, the common hepatic and common bile ducts appeared normal in caliber and contour, and the main pancreatic duct appeared normal. A ^{99m}Tc-sulfur colloid liver-spleen scan demonstrated nonhomogeneous activity throughout the slightly enlarged liver. The spleen was slightly enlarged, with relatively increased uptake. No marrow uptake was identified. Selective superior

mesenteric and hepatic angiography disclosed that the hepatic artery was a branch of the superior mesenteric artery; the major branches of the superior mesenteric artery were patent, and films obtained during the venous phase were normal. There was a slightly nonhomogeneous hepatographic phase suggestive of dilatation of the biliary radicles. No tumor blush or abnormal vascularity was observed, and the portal vein was patent. Fine-needle transhepatic cholangiography revealed multifocal concentric narrowing of major hepatic ducts in the region of the porta hepatis, with dilatation of secondary intrahepatic radicles. Combining the findings from the retrograde study, the scan, and angiography led to the conclusion that there was a small mass in the porta hepatis above the neck of the gallbladder. Percutaneous transhepatic cholangiography was the most diagnostic, showing the lesion from two sides. The preoperative diagnosis was an obstructing carcinoma at the porta hepatis, probably a cholangiocarcinoma. At exploration, a small tumor involving the proximal portion of the common hepatic duct and extending up into the liver was found. Frozen-section examination of the wedge-biopsy specimen revealed cords of tumor cells that had formed tubules with mucin secretion. Cholestasis and bile duct proliferation were observed in the adjacent liver. The anatomical diagnosis was adenocarcinoma of the hepatic ducts at the junction with the common hepatic duct.

9186 BILE DUCT INJURIES FROM NON-PENETRATING ABDOMINAL TRAUMA IN CHILDHOOD. (Eng.) Ahmed, S. (Adelaide Children's Hosp., 72 King William Rd., North Adelaide, South Australia 5006, Australia). *Aust. N.Z. J. Surg.* 46(3):209-212; 1976.

The cases of three children with bile duct injuries from nonpenetrating abdominal trauma are presented. The first patient, a 2.5-yr-old girl, was admitted with vomiting of bile-stained fluid. She was pale and dehydrated, with multiple bruises, and was thought to be a battered baby. Abdominal signs were equivocal, and initially she was managed conservatively, but on the 3rd day of hospitalization, she had signs of peritonitis. Surgical exploration revealed peritonitis and transection of the common bile duct. Continuity was established by performing a choledochoduodenostomy over a T-tube inserted through the common bile duct. The end of the transected duct was sutured to the duodenal mucosa. A drain was placed to the site of anastomosis, and the wound was closed. The second patient was a 6-yr-old boy who presented with upper abdominal pain, vomiting, increased pulse rate, and a blood pressure of 90/55 mm Hg after being hit by a car. Abdominal tenderness, rigidity, and absent bowel sounds were observed. At emergency laparotomy, a laceration of the inferior surface of the liver and transection of the common bile duct, which was identical with that in the first patient, were discovered. Choledochoduodenostomy was performed, and the surgical technique described in the first patient was adopted; this time, however, the T-tube was inserted through the duodenotomy. The third patient, a 5-yr-old girl who had been hit by a truck, presented with abdominal tenderness, rigidity, and absent bowel sounds. At surgery, a liver laceration and perforation in the common hepatic duct were found. The duct was not sutured, and a large drain was left *in situ*. All patients made satisfactory recoveries. Bile duct injuries due to nonpenetrating trauma present urgently when associated with liver laceration or other abdominal visceral injuries. When the duct injury is an isolated one, the ensuing clinical picture may be insidious. Surgical management is aimed at early recognition of the injury, followed by reconstruction of the biliary tract.

9187 RESULTS OBTAINED WITH AND INDICATIONS OF CHENODEOXYCHOLIC ACID IN THE TREATMENT OF CHOLELITHIASIS [Abstract]. (Fre.) Metreau, J. M.; Dhumeaux, D. (Hopital Henri-Mondor, 94010 Creteil, France). *Therapie* 32(4):407; 1977.

9188 COOPERATIVE TRIAL OF CHENODEOXYCHOLIC ACID APPLIED IN DIFFERENT DOSES IN CHOLESTEROL CHOLELITHIASIS. (Fre.) Bermann, C.; Betourne, C.; Brette, R.; Chaput, J. C.; Debray, C.; Etienne, J. P.; *et al.* (No affiliation given). *Gastroenterol. Clin. Biol.* 1(11):960; 1977.

9189 TREATMENT OF CHOLESTEROL CHOLELITHIASIS WITH β-SITOSTEROL: CONTROLLED THERAPEUTIC TRIAL. (Fre.) Marteau, C.; Reynier, M. O.; Mathieu, S.; Mule, A.; Crotte, C.; Gerolami, A.; Sarles, H. (Unite de Recherches de Pathologie Digestive, INSERM, U-31, 46, chemin de la Gaye, 13009 Marseille, France). *Gastroenterol. Clin. Biol.* 1(8/9):716; 1977.

9190 DIVERTICULUM OF THE COMMON BILE DUCT. (Ger.) Schwegler, N. (Kantonsspital Basel, Basel, Switzerland). *Fortschr. Geb. Roentgenstr. Nuklearmed.* 128(2):235-236; 1978.

9191 THE "SHORT" COMMON BILE DUCT: AN ATTEMPT AT SYSTEMATIZATION. (Fre.) Salembier, Y. (Cite Hospitaliere, 3e Est, F 59000 Lille, France). *Nouv. Presse Med.* 6(33):2977-2984, *passim*; 1977.

9192 GALLBLADDER INSERTED IN THE COMMON HEPATIC DUCT: A RARE ANOMALY OF THE BILE DUCTS (Ger.) Zimmermann, H. G. (Allgemeines Krankenhaus Hamburg-Barmbeck, Rubenkamp 148, D-2000 Hamburg 60, W. Germany). *Chirurg* 48(2):73-76; 1977.

9193 USE OF TETRACYCLINES IN BILIARY DISEASES: ASSESSMENT BY THE DETERMINATION OF SERUM AND TISSUE LEVELS. (Ger.) Pelz, K.; Herdter, F.; Marcushen, M. (Klinikum der Albert-Ludwigs-Universitat, Postfach 820, 7800 Freiburg i. Brsg., W. Germany). *Therapiewoche* 27(47):8585-8591, *passim*; 1977.

9194 CHOLERETIC ACTIVITY OF PROBILIN® IN MATCHED DOUBLE-BLIND TRIAL AFTER CHOLECYS-

TECTOMY. (Ger.) Mestrovic, N. J. (St. Markus-Krankenhaus, 5300 Bonn-Bad Godesberg, W. Germany). *Therapiewoche* 27(35):6077-6083, *passim*; 1977.

9195 MANAGEMENT OF HIGH OBSTRUCTIONS OF THE BILE DUCTS BY THE RODNEY-SMITH METHOD. (Ger.) Botticher, R.; Gauger, U. (Chirurgische Universitatsklinik, Krankenhausstrasse 12, D-8520 Erlangen, W. Germany). *Chirurg* 48(7):444-447; 1977.

9196 ERRORS IN BILE DUCT SURGERY. (Rus.) Avdei, L. V.; Matuzov, L. Z. (Second Clinic Hosp. Surgery, Minsk Medical Inst., Minsk, USSR). *Khirurgiia (Mosk.)* (7):134-138; 1977.

9197 RESULTS OF A NONCONTROLLED COOPERATIVE TRIAL OF CHENODEOXYCHOLIC ACID IN THE TREATMENT OF CHOLELITHIASIS. (Fre.) Bernades, P.; Bonfils, S.; Bourel, M.; Caulin, C.; Erlinger, S.; Ferrier, J. P.; *et al.* (No affiliation given). *Therapie* 32(4):405; 1977.

9198 GREY SCALE ULTRASONOGRAPHY: EVALUATING THE JAUNDICED PATIENT. (Eng.) Zusmer, N. R.; Harwood, S. J.; Pevsner, N. H.; Janowitz, W. R.; Serafini, A. N. (Univ. Miami Sch. Medicine, P.O. Box 520875, Miami, FL 33152). *South. Med. J.* 71(5):498-501; 1978.

9199 PERFORATED CHOLEDOCHAL CYST IN CHILDREN. (Eng.) Todani, T.; Tabuchi, K.; Watanabe, Y.; Nabeyama, A.; Emoto, T.; Munetomo, Y. (Okayama Univ. Medical Sch., 2-5-1 Shikatacho, Okayama City 700, Japan). *Z. Kinderchir.* 23(3):280-286; 1978.

9200 THE VALUE OF GRAY SCALE ULTRASOUND IN THE DIFFERENTIAL DIAGNOSIS OF SURGICAL AND NONSURGICAL JAUNDICE. (Eng.) Sabel, J. S.; Graham, D. Y.; Davis, R. E.; Malini, S. (Ben Taub General Hosp., Houston, TX). *Am. J. Gastroenterol.* 69(2): 149-153; 1978.

9201 VALUE OF OPERATIVE CHOLANGIOGRAPHY. (Eng.) Chatterjee, D. K.; Jones, W. M. (Royal Infirmary, Sheffield, England). *Br. J. Clin. Pract.* 32(4):105-106; 1978.

9202 NONOPERATIVE REMOVAL OF RETAINED COMMON-BILE-DUCT STONES. (Eng.) Ho, C. S. (Toronto General Hosp., Toronto, Ontario M5G 1L7, Canada). *Can. J. Surg.* 21(3):244-247; 1978.

9203 CHOLELITHIASIS AND ACOUSTIC SHADOWING. (Eng.) Grossman, M. (Univ. California, Irvine Medical Center, 101 City Drive South, Orange, CA 92668). *J. Clin. Ultrasound* 6(3):182-184; 1978.

9204 NON-OPERATIVE REMOVAL OF RETAINED STONES IN THE COMMON BILE DUCT. (Eng.) Rees, B. I.; Lawrie, B. W. (Dept. Surgery, Univ. South Wales, Wales). *Br. J. Surg.* 65(5):334-336; 1978.

9205 BILE COMPOSITION AND DEPOSITION OF CALCIUM CARBONATE IN GALLSTONES [Abstract]. (Eng.) Sutor, D. J.; Wilkie, L. I.; Clark, C. G. (Dept. Chemistry, Univ. Coll. London, London, England). *Gut* 18(5):A402; 1977.

9206 CHOLEDOCHOSCOPY IN DISEASES OF THE SPHINCTER OF ODDI. (Eng.) Vosse, A. *In: The Sphincter of Oddi. Proceedings of the Third Gastrointestinal Symposium, Nice, June 8-9, 1976.* (Basel: S. Karger): 120-122; 1977.

9207 PEROPERATIVE ENDOSCOPIC EVALUATION OF THE DISTAL BILE DUCT. (Eng.) Fourtanier, G.; Escat, J.; Lacroix, A. *In: The Sphincter of Oddi. Proceedings of the Third Gastrointestinal Symposium, Nice, June 8-9, 1976.* (Basel: S. Karger): 43-44; 1977.

See also, 8713, 8722, 8784, 8785, 8787, 8792, 8793, 8794, 8800, 8801, 8809, 8813, 8815, 8816, 8860, 8879, 8907, 8944, 9038, 9063, 9066, 9067, 9071, 9075, 9103, 9244.

GENERAL

9208 VERNER-MORRISON SYNDROME: CLINICAL STUDY. (Fre.) Dupuy, R. (Hopital Beaujon, 100, av. du General-Leclerc, 92110 Clichy, France). *Ann. Gastroenterol. Hepatol.* 13(6):599-605; 1977.

The Verner-Morrison (V-M) syndrome that accompanies a tumor of the Langerhans' cells of the pancreas is reviewed. The tumor is the rarest of the pancreatic tumors and, since a report of 64 cases published in 1974, not many more have been added to the literature. The average age of patients with this tumor is 47 yr with an age range of 17-72 yr. The tumor secretes a hormonal factor that induces intestinal peristalsis, causing symptoms of abundant, aqueous diarrhea. The diarrhea is accompanied by hypokalemia of 3.6-1.2 mEq and hypochlorhydria in 50% of the patients. Differential diagnosis of the V-M syndrome from Zollinger-Ellison disease is established by the ulcer and hyperchlorhydria in the latter and from carcinoids by the hypotension

and severe flushing of the skin in the latter. In 33-35% of the V-M syndrome patients, a malignant tumor is responsible for the disease, and prognosis is poor; the cause of the V-M syndrome is a benign adenoma of the pancreas in 41% of the patients and pancreatic hyperplasia in the remainder. Absence of a malignant tumor is not an assurance of a good prognosis, since one-third of the patients with adenomas die of renal insufficiency related to hypokalemia. Early diagnosis and surgical excision of a malignant tumor or subtotal pancreatectomy with restoration of the electrolyte balance with a benign tumor or hyperplasia are the recommended treatments.

9209 VERNER-MORRISON SYNDROME: MORPHOLOGICAL DATA DERIVED FROM HISTOENZYMOLOGY AND HISTOIMMUNOLOGY. (Fre.) Martin, E. D.; Galain, A.; Scotto, J.; Leclerc, J. P.; Ganter, P. (Hopital Antoine-Beclere, 157, rue de la Porte-de-Trivaux, 92141 Clamart, France). *Ann. Gastroenterol. Hepatol.* 13(6):615-627; 1977.

On the basis of 91 cases of the Verner-Morrison (V-M) syndrome (4 new cases, 87 from the literature), the epidemiological, histological, physiological, and immunological aspects of the syndrome are reviewed. Both sexes are equally likely to develop the V-M syndrome; the age range is 17-78 yr with an average age of 46 yr. The V-M syndrome is caused by a tumor of the pancreas that is larger than 3 cm in 78% of the patients. The tumor is malignant in 62.5% of the patients; otherwise the syndrome may be induced by an adenoma, polyadenomatosis, or hyperplasia of the pancreas. No ectopic tumors have been identified. Criteria for malignancy are not based on cytological characteristics since the tumor cells are almost always regular with little evidence of mitoses; it is based on the presence of intravascular emboli and hepatic or lymph node metastases. Ultrastructural studies, performed in 17 cases reported in the literature and in 3 in the present series, show two populations of secretory granules: one about 300 nm in diameter and the other about 625 nm. Histochemical immunofluorescence studies reveal the presence of a vasoactive intestinal polypeptide (VIP) that has also been found in bronchial carcinomas, a pheochromocytoma, and two ganglioneuroblastomas. In 50% of tumors in V-M patients, another hormone, the human pancreatic hormone (HPP), has been found associated with VIP. The presence of VIP in tumors known to be of nerve tissue origin may be evidence for the neuroectodermal origin of the tumor cells that cause the V-M syndrome.

9210 SECRETIN AND THE ZOLLINGER-ELLISON SYNDROME: RELIABILITY OF SECRETIN TESTS AND PATHOGENETIC ROLE OF SECRETIN. (Eng.) Stage, J. G.; Stadil, F.; Rehfeld, J. F.; Fahrenkrug, J.; Schaffalitzky de Muckadell, O. B. (H. C. Andersens Blvd. 47, 1553 Copenhagen V, Denmark). *Scand. J. Gastroenterol.* 13(4):501-511; 1978.

To establish the reliability of secretin tests in the diagnosis of the Zollinger-Ellison syndrome (ZE), the effects of an i.v. infusion (1 clinical U (CU)/kg/hr) or injection (1 CU/kg) of secretin on serum gastrin concentrations were compared in 25 hypersecretors with verified ZE and in 19 patients with duodenal ulcer disease. Secretin infusion raised the serum gastrin levels in the ZE patients. The increase appeared within 15 min and lasted until termination of the infusion. The peak value of 128 ± 13.1% of basal ($p=0.01$) was reached after 30 min. In ulcer patients, the serum gastrin concentration increased only after termination of the infusion (to 120.5 ± 8.5% of the basal level, $p<0.05$). Differences in the serum gastrin level during infusion between the ZE and ulcer patients were not significant, but the levels 5 min after an i.v. bolus injection were significantly different (238 ± 40% and 132 ± 12% of the basal level in ZE and ulcer patients, $p<0.01$ and $p<0.05$, resp.). However, not all ZE patients responded to the injection, and there were overlapping results in the two groups of patients both after infusion and after bolus injection. Pre- and postoperative studies showed that total gastrectomy did not influence either the gastrin response to secretin or the serum gastrin level. Serum gastrin corresponded to the four gastrin components I-IV. No changes in the gastrin component pattern occurred after secretin administration in any patient. The serum secretin concentrations in 11 fasting unoperated patients and 5 gastrectomized ZE patients tested (5.6 ± 0.75 and 2.2 ± 0.8 pM, resp.) were higher than in 74 normal subjects (1.4 ± 0.2 pM, $p<0.001$). This result does not support the hypothesis that secretin release by acid entering the duodenum drives the gastrin release from gastrinomas, since exogenous secretin often failed to raise the serum gastrin concentration and since serum gastrin concentrations did not decrease after total gastrectomy. Because of the failure of some ZE patients to respond to the secretin test, a positive result is not sufficient to establish the diagnosis of ZE. The secretin test appears to have limited diagnostic value in ZE patients.

9211 REPORT ON THE UNITED STATES EXPERIENCE WITH CIMETIDINE IN ZOLLINGER-ELLISON SYNDROME AND OTHER HYPERSECRETORY STATES. (Eng.) McCarthy, D. M. (Natl. Inst. Arthritis, Metabolism and Digestive Diseases, Bldg. 10, Rm. 9D-15, Natl. Inst. Health, Bethesda, MD 20014). *Gastroenterology* 74(2, Part 2):453-458; 1978.

Experience with cimetidine in the treatment of 61 patients with the Zollinger-Ellison syndrome, encountered over a 2-yr period, is presented. Half the patients have been treated for over 1 yr. Two-thirds of the patients responded to 300 mg of the drug p.o. every 6 hr; others required up to 600 mg every 6 hr. In adequate doses, the drug was highly effective; it controlled pain and dyspepsia, restored weight, abolished diarrhea, and allowed healing of ulcers and other inflammatory conditions. Missed or reduced doses led to rapid return of symptoms. Progression of the basic neoplastic process, with associated secretory drive, was unimpeded. Patient acceptance of the drug was 100%,

and apart from minor transient abnormalities, gynecomastia (5 cases) and liver dysfunction (3 cases), which resolved while treatment continued, no serious adverse effects were seen. Of 61 patients, 48 are still on the drug, 3 who were well controlled were also treated surgically, 5 died for reasons unrelated to therapy, and 5 had significant problems. The drug provides an alternative to total gastrectomy and can be recommended with confidence for the suitably selected patient.

9212 HISTOLOGICAL, HISTOENZYMOLOGICAL AND ULTRAMICROSCOPIC ASPECTS OF PANCREATIC TUMORS IN ZOLLINGER-ELLISON SYNDROME. (Fre.) Potet, F. (Hopital Beaujon, 100, bd du General-Leclerc, 92110 Clichy, France). *Ann. Gastroenterol. Hepatol.* 13(6):591-597; 1977.

Histological, histoenzymological, and ultramicroscopic features of the pancreatic tumors causing the Zollinger-Ellison (ZE) syndrome are discussed. The histochemical behavior of the tumor does not always parallel that of the gastrin cell from which it has been thought to arise. Characteristically, the gastrin cell shows argyrophilia by the methods of Grimelius and Sevier-Munger, fluorescence when exposed to formol, metachromasia with toluene blue at pH 5.5, and hematoxyline reaction with lead. The reaction found most consistently is argyrophilia, which identifies tumor cells with an endocrine origin. Ultrastructurally, the granulation typical of the gastrin cell is not observed in the pancreatic tumors. Unlike the normal gastrin cell, granules in the tumor cells are of variable density, irregular size, and not abundant. Immunochemically, the tumor cells react like the gastrin cells, since they react with antiserum marked with the isothiocyanate of fluorescein (indicating the presence of gastrin). In 20% of cases, there are two, three, or more types of secretions present, but secretion of gastrin predominates. The histogenesis of the tumors that cause the ZE syndrome is not well understood.

9213 NEW DATA ON METHODS FOR MORPHOLOGICAL EXAMINATION OF PANCREATIC GROWTHS IN ZOLLINGER-ELLISON SYNDROME. (Fre.) LaMarque, J. L.; Bruel, J. M.; Dondelinger, R.; Polge, D. (Hopital Saint-Eloi, 34059 Montpellier Cedex, France). *Ann. Gastroenterol. Hepatol.* 13(6):579-590; 1977.

Diagnosis by conventional x-ray and angiographic examinations of the pancreatic tumors that cause the Zollinger-Ellison (ZE) syndrome is discussed. The cause of the ZE syndrome is a small pancreatic tumor, often multiple, sometimes reduced to simple hyperplasia. The criteria for malignancy of these tumors is the presence of hepatic metastases. The metastases are highly vascular and can be readily diagnosed by selective angiography of the splenic and superior mesenteric veins. In the absence of hepatic metastases, the pancreatic tumors can be identified more readily if they are highly vascular. Selective arteriography of the gastroduodenal and dorsal pancreatic arteries will show the hypervascular pancreatic tumors. In the 20% of pancreatic tumors that are not highly vascular, diagnosis must be made by indirect signs such as compression of arteries and veins and lacunar x-ray images of the body of the pancreas. Usually, only large tumors will produce these signs. The use of sonography, scintigraphy, and tomodensitometry in the diagnosis of pancreatic tumors is also discussed.

9214 VIRUSES AND DIARRHEA--A REVIEW. (Eng.) Steinhoff, M. C. (Univ. Rochester Medical Center, 601 Elmwood Ave., Rochester, NY 14642). *Am. J. Dis. Child.* 132(3):302-307; 1978.

9215 RADIATION THERAPY IN THE TREATMENT OF MALIGNANT SALIVARY GLAND TUMORS. (Eng.) Elkon, D.; Colman, M.; Hendrickson, F. R. (Rush-Presbyterian-St. Luke's Medical Center, 1753 W. Congress Parkway, Chicago, IL 60612). *Cancer* 41(2):502-506; 1978.

9216 ACUTE FOLATE DEFICIENCY ASSOCIATED WITH INTRAVENOUS NUTRITION WITH AMINOACID-SORBITOL-ETHANOL: PROPHYLAXIS WITH INTRAVENOUS FOLIC ACID. (Eng.) Wardrop, C. A. J.; Lewis, M. H.; Tennant, G. B.; Williams, R. H. P.; Hughes, L. E. (Welsh Natl. Sch. Medicine, Heath Park, Cardiff CF4 4XW, Wales). *Br. J. Haematol.* 37(4):521-526; 1977.

9217 DRUG KINETICS AND METABOLISM IN PEM. (Eng.) Anonymous. (No affiliation given). *S. Afr. Med. J.* 53(9):329-330; 1978.

9218 ACQUIRED EVENTRATION OF THE DIAPHRAGM--RESULTS OF SURGERY. (Eng.) Varpela, E.; Laustela, E. V.; Viljanen, A. (Univ. Central Hosp., SF-00290 Helsinki 29, Finland). *Ann. Chir. Gynaecol.* 66(6):284-289; 1977.

9219 FASTING METABOLISM IN INFANTS. I. EFFECT OF SEVERE UNDERNUTRITION ON ENERGY AND PROTEIN UTILIZATION. (Eng.) Kerr, D. S.; Stevens, M. C. G.; Robinson, H. M. (Rainbow Babies and Childrens Hosp., 2101 Adelbert Rd., Cleveland, OH 44106). *Metabolism* 27(4):411-435; 1978.

9220 ACINIC CELL CARCINOMA OF SALIVARY ORIGIN: A CLINICOPATHOLOGIC STUDY OF 67 CASES. (Eng.) Spiro, R. H.; Huvos, A. G.; Strong, E. W. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021). *Cancer* 41(3):924-935; 1978.

9221 ENTEROTOXIN-PRODUCING Escherichia coli AND DIARRHOEA AMONG NEWBORN BABIES IN DARWIN. (Eng.) Luke, R. K. J.; Ryan, N. J.; Barnes, G. L.; Bishop, R. F.; Dayaratne, R. D. B. (Sch.

Agriculture, La Trobe Univ., Bundoora, Victoria 3083, Australia). *Med. J. Aust.* 1(3):170-171; 1977.

9222 PARVOVIRUS GASTROENTERITIS--A NEW ENTITY FOR AUSTRALIA. (Eng.) Christopher, P. J.; Grohmann, G. S.; Millsom, R. H.; Murphy, A. M. (Western Metropolitan Health Region, New South Wales, Australia). *Med. J. Aust.* 1(3):121-124; 1978.

9223 CHRONIC DIARRHOEAS IN ADULTS. (Eng.) Chatterjee, H. (Sch. Tropical Medicine, Calcutta, India). *J. Indian Med. Assoc.* 69(11):259-261; 1977.

9224 PLASMA VIP IN PATIENTS WITH WATERY DIARRHEA SYNDROME. (Eng.) Gardner, J. D. (Natl. Inst. Health, Bethesda, MD). *Am. J. Dig. Dis.* 23 (4):370-373; 1978.

9225 AMYLASE ELEVATION ATTRIBUTABLE TO AN OVARIAN NEOPLASM. (Eng.) Corlette, M. B.; Dratch, M.; Sorger, K. (Harvard Medical Sch., Boston, MA). *Gastroenterology* 74(5, Part 1):907-909; 1978.

9226 DISEASES AND DRUG PROTEIN BINDING. (Eng.) Tillement, J. P.; Lhoste, F.; Giudicelli, J. F. (Faculty Medicine, Univ. Paris Sud, 8 Rue du General-Sarrail, 94010 Creteil, France). *Clin. Pharmacokinet.* 3(2):144-154; 1978.

9227 HUMAN MILK AND GUT INFECTION IN THE NEWBORN. (Eng.) Bullen, J. J. (Natl. Inst. Medical Res., London, England). *J. Appl. Med.* 4(3):193-197; 1978.

9228 ACUTE GASTROENTERITIS AMONG SCHOOLCHILDREN ASSOCIATED WITH REOVIRUS-LIKE AGENT. (Eng.) Hara, M.; Mukoyama, J.; Tsuruhara, T.; Ashiwara, Y.; Saito, Y.; Tagaya, I. (Natl. Inst. Health, Musashimurayama, Tokyo 190-12, Japan). *Am. J. Epidemiol.* 107(2):161-169; 1978.

9229 ZINC DEFICIENCY DURING TOTAL PARENTERAL NUTRITION IN CHILDHOOD. (Eng.) Suita, S.; Ikeda, K.; Nagasaki, A.; Hayashida, Y. (Kyushu Univ. Hosp., 1-1, 3-chome, Maedashi, Higashi-ku, Fukuoka, Japan). *J. Pediatr. Surg.* 13(1):5-9; 1978.

9230 GASTROSCHISIS: NO MYTH. (Eng.) Noordijk, J. A.; Bloemsma-Jonkman, F. (Sophia Children's Hosp., Gordelweg 160, Rotterdam, Netherlands). *J. Pediatr. Surg.* 13(1):47-49; 1978.

9231 ENVIRONMENTAL FACTORS AND THE DEVELOPMENT OF DISEASE AND INJURY IN THE ALIMENTARY TRACT. (Eng.) Schedl, H. P. (Univ. Iowa Coll. Medicine, Iowa City, IA 52242). *Environ. Health Perspect.* 20:39-54; 1977.

9232 THE PATHOGENESIS OF DERMATITIS HERPETIFORMIS. (Eng.) Katz, S. I.; Strober, W. (Natl. Cancer Inst., Building 10, Room 12N238, Natl. Inst. Health, Bethesda, MD 20014). *J. Invest. Dermatol.* 70(2):63-75; 1978.

9233 ETHANOL AND DEVELOPMENT OF DISEASE AND INJURY TO THE ALIMENTARY TRACT. (Eng.) Krawitt, E. L. (Coll. Medicine, Univ. Vermont, Burlington, VT 05401). *Environ. Health Perspect.* 20:71-73; 1977.

9234 TREATMENT WITH H_2 RECEPTOR ANTAGONISTS: ZOLLINGER ELLISON SYNDROME. (Eng.) Siepler, J. K.; Donahue, P.; Campagna, K.; Bombeck, C. T. (Univ. Illinois Coll. Pharmacy, Urbana, IL 61801). *Ill. Med. J.* 153(4):283-287; 1978.

9235 PHLEGMONOUS ENTEROCOLITIS. (Eng.) Rosen, Y.; Won, O. H. (Kings County Hosp. Center, 451 Clarkson Ave., Brooklyn, NY 11203). *Am. J. Dig. Dis.* 23(3):248-256; 1978.

9236 A STUDY OF ETIOLOGIC AGENTS IN INFANTILE GASTROENTERITIS. (Eng.) Mundel, G.; Kohn, A.; Yaron, R.; Strossman, Y. (Asaf Harofe Government Hosp., Zerifin, Israel). *Isr. J. Med. Sci.* 14(3):391-394; 1978.

9237 CLINICAL COMPARISON OF CALCIUM POLYCARBOPHIL AND KAOLIN-PECTIN SUSPENSIONS IN THE TREATMENT OF ACUTE CHILDHOOD DIARRHEA. (Eng.) Rutledge, M. L.; Willner, M. M.; Clayton, J. M. (P.O. Box 377, Memphis, TN 38151). *Curr. Ther. Res.* 23(4, Part 1):443-447; 1978.

9238 EVALUATION OF GASTROINTESTINAL MICROFLORA IN NECROTIZING ENTEROCOLITIS. (Eng.) Bell, M. J.; Feigin, R. D.; Ternberg, J. L.; Brotherton, T. (St. Louis Children's Hosp., 500 S. Kingshighway, St. Louis, MO 63110). *J. Pediatr.* 92(4):589-592; 1978.

9239 TRANSIENT STEATORRHEA FOLLOWING EPISODES OF MILD DIARRHEA IN EARLY INFANCY. (Eng.) MacLean, W. C., Jr.; Klein, G. L.; Lopez de Romana, G.; Massa, E.; Graham, G. G. (Sch. Hygiene and Public Health, Johns Hopkins Univ., Baltimore, MD). *J. Pediatr.* 92(4):562-565; 1978.

9240 EOSINOPHILIC GASTROENTERITIS WITH ASCITES AND COLON INVOLVEMENT. (Eng.) Levinson, J. D.; Ramanathan, V. R.; Nozick, J. H. (New Jersey Coll. Medicine, Newark, NJ). *Am. J. Gastroenterol.* 68(6):603-607; 1977.

9241 ACRODERMATITIS ENTEROPATHICA--A ZINC DEFICIENCY STATE. (Eng.) Rubin, I. L.; Hansen, J. D. L.; Goldberg, B.; Presbury, D. G. C.; Kilroe-Smith, T. A. (Dept. Pediatrics, Univ. Witwatersrand, Johannesburg, South Africa). *S. Afr. Med. J.* 53(13):497-498; 1978.

9242 ENDOSCOPIC PHOTOCOAGULATION BY LASER IRRADIATION IN THE GASTROINTESTINAL TRACT OF MAN. (Eng.) Fruhmorgen, P.; Bodem, F.; Reidenbach, H. D.; Kaduk, B.; Demling, L. (Medizinischen Univ.-Klinik mit Poliklinik, Krankenhausstrasse 12, D-8520 Erlangen, W. Germany). *Acta Hepatogastroenterol.* 25(1):1-5; 1978.

See also, 8632, 8778, 8799, 8806, 8812, 8818, 8820, 8876, 8879, 8893, 8933, 8949, 8992, 9001, 9009, 9027, 9045, 9057, 9108, 9263.

GENERAL

Parasitology

9243 AMEBIASIS: EPIDEMIOLOGIC STUDIES IN THE UNITED STATES, 1971-1974. (Eng.) Krogstad, D. J.; Spencer, H. C., Jr.; Healy, G. R.; Gleason, N. N.; Sexton, D. J.; Herron, C. A. (Center for Disease Control, Atlanta, GA). *Ann. Intern. Med.* 88(1):89-97; 1978.

Seven investigations of suspected foci of amebiasis in the United States that were performed by the Center for Disease Control between October 1971 and June 1974 indicated that the disease was being vastly overdiagnosed by a number of laboratories. The problem encountered most frequently in these investigations was that WBC in the stool were identified as *Entamoeba histolytica*. Two laboratories found to be in error were in community hospitals, and one was at a teaching hospital associated with a medical school and a school of public health. Altogether, these three laboratories had been diagnosing about 1,200 cases of amebiasis a year for 20 yr. When amebiasis does occur, it is likely to be misdiagnosed. In one outbreak involving four cases and three deaths, amebiasis was not diagnosed until two patients had died and another was critically ill. Sporadic cases may be mistakenly diagnosed as ulcerative colitis and inappropriately treated with steroids. Stool examination for intestinal protozoa is difficult and requires a highly trained laboratory technologist. Because of this, serology should play an increasingly important role by detecting positive titers in patients with active intestinal amebiasis who might otherwise be treated for nonspecific ulcerative colitis. Endemic foci of amebiasis continue to exist in the United States as revealed by two of the investigations, which indicated that the disease was associated with poor sanitation and was transmitted by a person-to-person mode.

9244 BILIARY ASCARIASIS PRESENTING IN THE UNITED STATES. (Eng.) Schulman, A. (Groote Schuur Hosp., Cape Town, South Africa). *Am. J. Gastroenterol.* 68(2):167-170; 1977.

The case of a 44-yr-old Korean woman who presented in the United States with biliary ascariasis is reported. Several weeks after leaving Korea she presented with upper abdominal pain, light-colored diarrhea, nausea, vomiting, weight loss, and fever. I.v. cholangiography and tomography revealed a common duct dilated to 17 mm and containing a long, vermiform filling defect, as well as a rounded defect just below it. Barium enema and upper gastrointestinal and small bowel examinations showed several similar vermiform defects in the upper jejunum and duodenum. A conventional 2-day regimen of p.o. peperazine was administered. A repeat i.v. cholangiogram 9 days after this treatment still showed the round defect in the common bile duct, but the vermiform filling defect had disappeared. Exploration and operative cholangiography were carried out during surgery for a gallstone and a stone in the common bile duct. No worms or other abnormalities were found. *Ascaris lumbricoides* can usually be visualized by i.v. cholangiography because of the absence of obstructive jaundice and can therefore be specifically treated by nonsurgical methods. The radiographic appearance is diagnostic.

9245 LIVER AFFECTION ASSOCIATED WITH *Yersinia enterocolytica* INFECTION. (Eng.) Saebo, A. (Univ. Bergen Dept. Surgery, N-5016 Haukeland sjukehus, Bergen, Norway). *Acta Chir. Scand.* 143 (7/8):445-450; 1977.

A systematic screening of patients with acute abdominal disease of unknown origin revealed several cases with a positive antibody titer to *Yersinia enterocolytica*, coincident with elevated serum levels of aspartate amino transferase (ASAT), lactate dehydrogenase (LD), alkaline phosphatase

(AP), and bilirubin. Two cases are presented in detail. A woman born in 1916 had had abdominal pain, fever, and diarrhea since childhood; at age 11 she had an erythema nodosum, and about 1940 she developed epidemic hepatitis of brief duration. Colics in the right side of the abdomen followed by meteorism, vomiting, and diarrhea lasting 2-3 days began in 1949. Plain radiographs showed peritoneal irritation and cholecystography was negative. Right upper quadrant pain, diarrhea, fever, bloody stools, a 5-kg weight loss, and swollen and painful joints led to the admission during which the positive *Y. enterocolitica* antibody titer was found. A liver biopsy revealed uncharacteristic liver changes, and splenomegaly was diagnosed scintigraphically. She was treated with sulfamethoxasol (1 g b.i.d.) and then ampicillin (0.5 g t.i.d.). The antibody titer (to *Y. enterocolytica* serogroup 3) remained positive but was stationary (1/160-1/320), and no pathogens were isolated from the stools. She was readmitted in 1977 for dark urine, clay-colored stools, and right-sided abdominal tenderness; i.v. cholecystography revealed no abnormalities. A woman born in 1900 had rheumatic fever in 1948 and was seen twice in 1974 (for uncharacteristic dyspepsia and for acute swelling of the left knee joint). In 1976, she was admitted for dark urine and clay-colored stools; exploratory laparotomy found no gallbladder disease but an inflamed liver. Biopsy revealed severe cholestasis, moderate fatty degeneration, inflammation, and single-cell necrosis. Prednisone therapy was instituted for 2 weeks, withdrawn, and reinstituted following relapse. Tetracycline was given because seroreactions to *Y. enterocolytica* gave titers of 1/10 and then 1/320 over a 1-week period; this titer continued to vary, improvement was very slow, and electrophoresis showed a chronic, active process. No pathogens were isolated from fecal samples. It is suggested that chronic and acute forms of liver disease caused by *Y. enterocolytica* exist and may resemble cholecystitis in presentation.

9246 THE RESULTS OF SURGERY IN AMEBIC LIVER ABSCESS: EXPERIENCES IN EIGHTY-THREE PATIENTS. (Eng.) Eggleston, E. C.; Verghese, M.; Handa, A. K.; Gill, S. S. (Christian Medical Coll., Ludhiana, Punjab 141001, India). *Surgery* 83(5): 536-539; 1978.

9247 PREVALENCE OF INTESTINAL PARASITES: (A RURAL STUDY). (Eng.) Yunus, M.; Zaheer, M.; Sinha, S. N. (Jawaharlal Nehru Medical Coll., Aligarh Muslim Univ., Aligarh, India). *J. Indian Med. Assoc.* 69(11):241-245; 1977.

9248 AMOEBIC LIVER ABSCESS. (Eng.) Habibullah, C. M.; Rao, S. R.; Rao, G. N.; Azam, M.; Ramachandran, A. (Osmania Medical Coll., Hyderabad, India). *J. Indian Med. Assoc.* 69(11):247-250; 1977.

9249 SCHISTOSOMIASIS IN IRRIGATION SCHEMES IN THE AWASH VALLEY, ETHIOPIA. (Eng.) Kloos, H.; Lemma, A. (George Williams Hooper Foundation, Univ. California, San Francisco, CA 94122). *Am. J. Trop. Med. Hyg.* 26(5, Part 1):899-908; 1977.

9250 EPIDEMIOLOGY AND MASS-TREATMENT OF ASCARIASIS IN SIX RURAL COMMUNITIES IN CENTRAL IRAN. (Eng.) Arfaa, F.; Ghadirian, E. (Sch. Public Health and Inst. Public Health Res., Univ. Tehran, P.O. Box 1310, Tehran, Iran). *Am. J. Trop. Med. Hyg.* 26(5, Part 1):866-871; 1977.

9251 TREATMENT OF SYMPTOMATIC INTESTINAL AMOEBIASIS WITH TINIDAZOLE. (Eng.) Garcia, E. G. (Inst. Public Health, Univ. Philippines System, Manila, Philippines). *Drugs* 15(Suppl. 1):16-18; 1978.

See also, 8738, 8739, 8781, 8808, 9104, 9115, 9214, 9223, 9236.

GENERAL

Regional Enteritis

9252 METRONIDAZOLE IN CROHN'S DISEASE: A DOUBLE BLIND CROSS-OVER CLINICAL TRIAL. (Eng.) Blichfeldt, P.; Blomhoff, J. P.; Myhre, E.; Gjone, E. (Rikshospitalet, Oslo 1, Norway). *Scand. J. Gastroenterol.* 13(1):123-127; 1978.

A double-blind crossover study of the value of metronidazole was carried out in 22 patients with active Crohn's disease who were also being treated with salazosulfapyridin or prednisone. The patients received metronidazole (1,000 mg/day, p.o.) or placebo for 2 months. No statistically significant effect was observed in the overall clinical condition of the 20 patients who completed the trial, although the hemoglobin level rose and ESR fell significantly ($p<0.01$ and $p<0.05$, resp.) during metronidazole treatment. In the six patients in whom Crohn's disease involved only the colon, there was a striking improvement in symptoms, which included diarrhea and abdominal pain. Hemoglobin

rose and ESR fell in these patients during treatment. These results indicate that metronidazole is useful only in the treatment of Crohn's colitis.

9253 THE LONG-TERM PROPHYLACTIC EFFECT OF SALAZOSULPHAPYRIDINE (SALAZOPYRIN®) IN PRIMARILY RESECTED PATIENTS WITH CROHN'S DISEASE: A CONTROLLED DOUBLE-BLIND TRIAL. (Eng.) Wenckert, A.; Kristensen, M.; Eklund, A. E.; Barany, F.; Jarnum, S.; Worning, H.; *et al.* (Herlev Univ. Hosp., DK 2730 Herlev, Denmark). *Scand. J. Gastroenterol.* 13(2):161-167; 1978.

A double-blind study of the possible prophylactic effect of salazosulphapyridine (SASP) in the treatment of Crohn's disease was carried out in 66 patients. Treatment with SASP (3 g/day, 32 patients) or placebo was started within 1 month following the patients' first surgical resection of diseased small or large bowel. The treatment period was 1 yr, and this was later prolonged to 18 months. Relapse-free patients were observed for 24 months. Using actuarial analysis, the four relapses that occurred in the patients treated with SASP and the nine in the placebo group after 18 months of treatment were calculated to correspond to cumulative relapse rates of 0.13 and 0.45, resp. ($0.10<p<0.15$). The results support the proposal that SASP is useful in preventing the recurrence of Crohn's disease following surgery. The need for large-scale trials is emphasized.

9254 URINARY OXALATE EXCRETION RELATED TO ILEOCOLIC SURGERY IN PATIENTS WITH CROHN'S DISEASE. (Eng.) Andersson, H.; Filipsson, S.; Hulten, L. (Sahlgrenska sjukhuset, S-413 45 Goteborg, Sweden). *Scand. J. Gastroenterol.* 13(4): 465-469; 1978.

The effect of high oxalate and high fat diets on urinary oxalate excretion in 63 patients with Crohn's disease was studied before and after surgery and compared with results in 26 healthy controls. The urinary oxalate excretion was studied in relation to the length of the resected ileal segment in patients who had restorative and colectomy procedures and also in relation to the fecal excretion of fat and bile salts and to urinary excretion of vitamin B_{12} and calcium. Subjects received a standard hospital diet (about 75 g fat), with or without a high oxalate (800-900 mg) supplement from spinach and/or a high fat (150 g fat) supplement. Urinary oxalate excretion increased significantly when a high-oxalate diet was consumed by 15 patients with ileal or ileocolic disease ($p<0.001$) and in 23 patients with ileocolic resection or colectomy ($p<0.005$) compared with oxalate excretion on the standard diet. Controls on standard and high-oxalate diets had nearly identical oxalate excretion levels. Urinary oxalate excretion was significantly higher than control values in patients with ileocolic resections on both diets ($p<0.02$ and $p<0.001$, resp.) and in colectomized patients on the high-oxalate diet ($p<0.001$). A high-fat/high-oxalate diet increased urinary oxalate excretion in 25 patients with ileal resection ($p<0.01$) but not in 11 colectomized patients. In 35 patients subjected to ileal resection in conjunction with a limited colon resection (but not in the colectomized patients), the urinary oxalate excretion increased with the length of the resected ileal segment ($r=0.40$, $p<0.02$). A linear correlation ($r=0.50$, $p<0.005$) was obtained between the excretion of fat and of urinary oxalate in 33 patients with ileocolic resection (but none was observed in the colectomized patients). In 34 patients subjected to ileocolic resection, there was an exponential correlation between urinary oxalate and vitamin B_{12} excretion ($r=0.51$, $p<0.005$). No correlation between urinary oxalate excretion and bile salt malabsorption was found. In all patients, calcium excretion was low (5 mmol/24 hr for most patients), but no correlation with urinary oxalate excretion was found. A low-fat, low-oxalate, and high-calcium diet is recommended to prevent hyperoxaluria in patients after ileocolic resection, particularly where a substantial part of the colon is preserved.

9255 INVESTIGATION OF THE PATHOGENESIS AND ETIOLOGY OF CROHN'S DISEASE. (Ger.) Raguse, T.; Bracker, H. P. (Abtlg. Chirurgie, Med. Fakultat d. RWTH, Goethestrasse 27/29, 51 Aachen, W. Germany). *Aktuel. Chir.* 12(5):317-326; 1977.

A study was undertaken to clarify the role of humoral and cellular competence of the immune system in the etiology and pathogenesis of Crohn's disease (CD). Nine women and 11 men with severe, chronic recurring CD were studied. The levels of complement factors C3 and C4 in the patients did not differ significantly from those of controls. IgG and IgA determinations were also within normal limits. IgM levels (222.1 ± 96.55 mg%), however, were significantly ($p<0.05$) above normal (102 ± 30 mg%). The sera of CD patients had a significant ($p<0.05$) inhibitory effect on stimulation in the mixed lymphocyte reaction (MLR). CD patients also had markedly increased lymphocyte counts. A marked increase in the absolute numbers of B cells existed, while the T cell count was decreased. Phytohemagglutinin stimulation showed a significant ($p<0.05$) anergy of the lymphocytes; in six patients, there was an extreme decrease in sensitivity to stimulation of the T lymphocytes. These findings indicate a distribution of the cellular immune system (in the sense of lymphocytic anergy) of undetermined cause. The inhibitory effect of CD sera in the MLR indicates blocking of the lymphocyte-specific cell function, which has been recognized in viral infections. Treatment of CD with immunosuppressive drugs is therefore questionable, while stimulation of the cellular immune system, as with Thymosin or Levamisole, is beneficial. Dietary supplements of amino acids are essential.

9256 PSEUDOPOLYPOID FORMS OF CROHN'S DISEASE. (Fre.) Rouger, P.; Tubiana, J. M.; Douvin, D.; Chermet, J.; Monnier, J. P.; Malafosse, M.; *et al.* (Hopital Saint-Antoine, 184, rue du Faubourg

Saint-Antoine, F 75571 Paris Cedex 12, France). *J. Radiol. Electrol. Med. Nucl.* 58(12):807-810; 1977.

Three cases are reported in men (ages 49, 34, and 39 yr, resp.) and one in a woman (age 26 yr) of Crohn's disease (regional ileitis) of a pseudopolypoid type. The inflammatory polyps were located in the colon and the ileum and could be localized or diffuse. Actually, the polyps were the result of deep ulcerations of the intestinal mucosa surrounding healthy or only edematous mucosa that appeared to project into the lumen of the intestinal tract. Diagnostically, pseudopolypoid Crohn's disease must be distinguished from ulcerohemorrhagic rectocolitis, familial intestinal polyposis, and lymphoid polyposis. X-ray studies alone cannot be relied upon for accurate diagnosis, which is established by histological examination of biopsy material.

9257 ISOLATED CROHN'S DISEASE OF VERMIFORM APPENDIX. (Eng.) Tseng, C. H.; Zinner, E.; Lahiri, N.; Das, K. (Veterans Admin. Center, Dayton, OH). *Abdom. Surg.* 20(1):17-19; 1978.

9258 SUBDIAPHRAGMATIC AIR AS A SIGN OF PERFORATED REGIONAL ENTERITIS: CASE REPORT. (Eng.) Welch, J. P. (Womack Army Hosp., Fort Bragg, NC 28307). *Milit. Med.* 142(11):880-881; 1977.

9259 RELATIONSHIP OF ERYTHEMA NODOSUM TO OTHER MANIFESTATIONS OF CROHN'S DISEASE. (Eng.) Jacobs, M.; Winkelman, E. I.; Farmer, R. G. (Cleveland Clinic Foundation, Cleveland, OH). *Cleve. Clin. Q.* 44(4):145-148; 1977.

9260 GIANT PSEUDOPOLYPOSIS CAUSING COLONIC OBSTRUCTION: REPORT OF A CASE SEEMINGLY ASSOCIATED WITH CROHN'S DISEASE OF THE COLON. (Eng.) Gruner, O. P. N.; Refsum, S.; Fausa, O.; Hognestad, J. (Rikshospitalet, Oslo 1, Norway). *Scand. J. Gastroenterol.* 13(1):65-69; 1978.

9261 LOCALIZED GIANT PSEUDOPOLYPOSIS IN CROHN'S DISEASE: COLONOSCOPIC FINDINGS. (Eng.) Shah, S. M.; Rogers, H. L.; Nagai, N. (Veterans Admin. Hosp., 300 E. Roosevelt Rd., Little Rock, AR 72206). *Dis. Colon Rectum* 21(2): 104-106; 1978.

9262 RADIOLOGY OF CROHN'S DISEASE. (Eng.) Bodart, P.; Pringot, J. (Cliniques Universitaires Saint-Luc, Universite Catholiques de Louvain, Avenue Hippocrate 10, 1200 Bruxelles, Belgium). *J. Belg. Radiol.* 60(1):83-101; 1977.

9263 ANOREXIA NERVOSA AND CROHN'S DISEASE. (Eng.) Metcalfe-Gibson, C. (Manchester Royal Infirmary, S5 Unit, Oxford Rd., Manchester, England). *Br. J. Surg.* 65(4):231-233; 1978.

See also, 8625, 8973, 9006, 9026, 9027, 9028, 9036, 9231.

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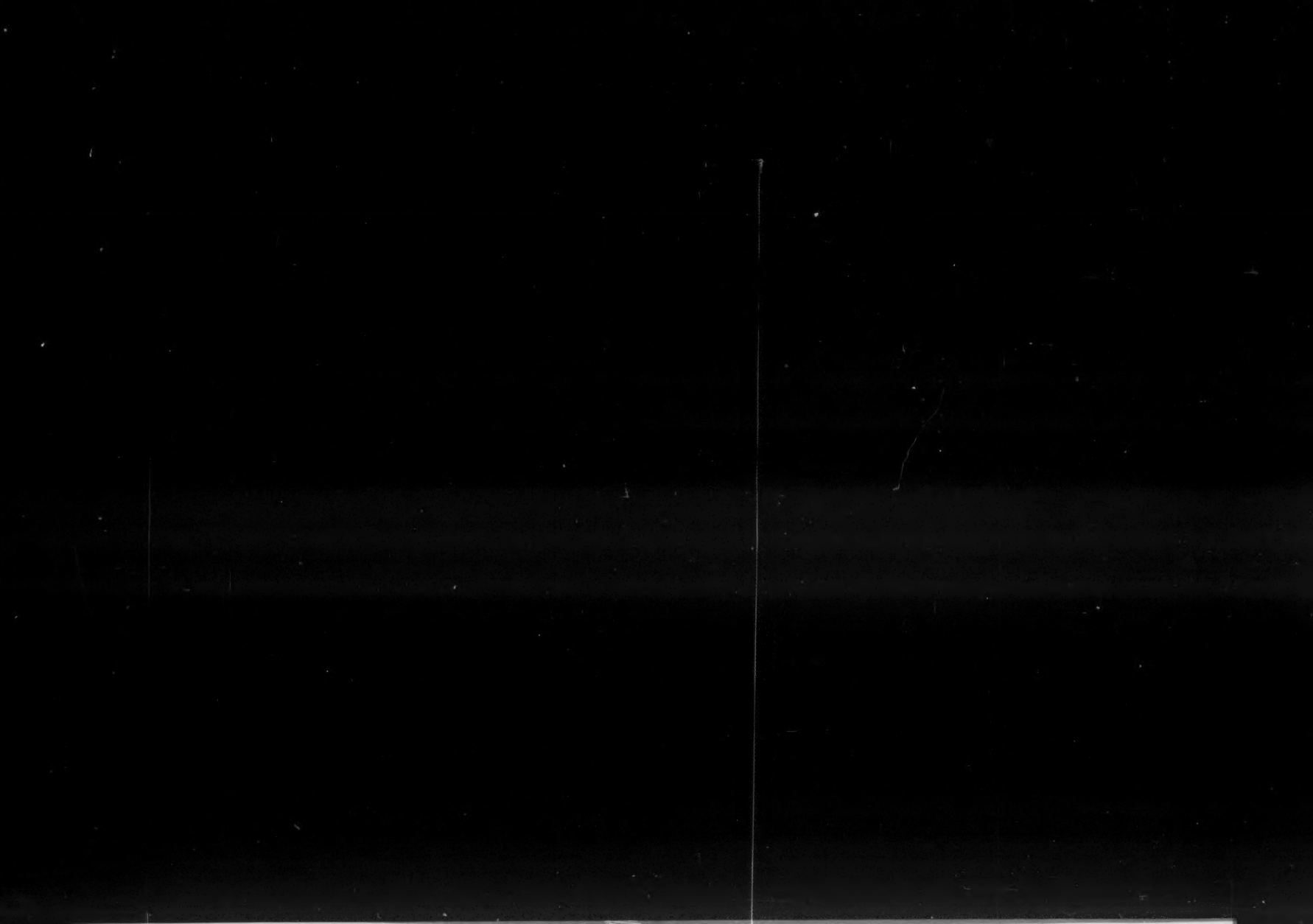
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